



NISHTAR
PUBLICATIONS (Pvt). Ltd.

**2ND SECOND
EDITION**

GRIP THE TOACS

IMM AND FCPS PART II MEDICINE

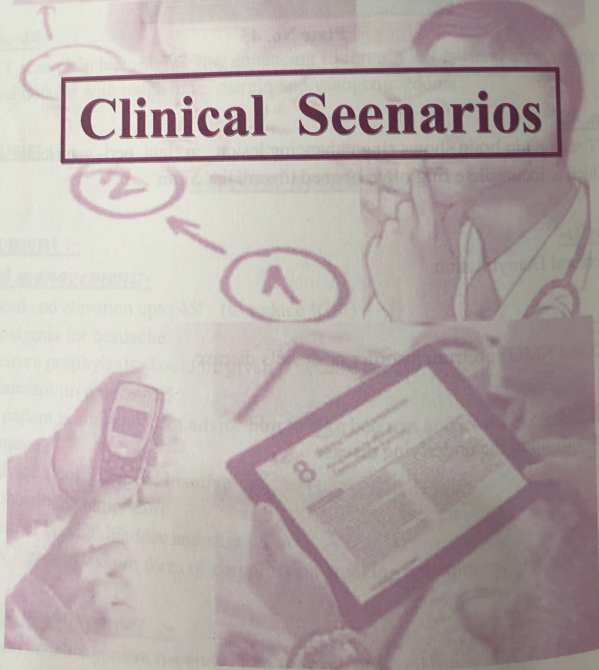
THE MOST TRUSTED BOOK FOR TOACS

Malik Samiullah Douna





Clinical Scenarios



Case 01:-

A 45 Year old male presented with the following blood report:-

- Serum bilirubin: 20 mg/dL (normal up to 1.2 mg/dL).
- SGPT: 80 U/L (normal <20 U/L)
- SGPT: 800 IU/L (normal <20 IU/L).
- Serum alkaline phosphatase: 1400 IU/L (normal 20-100 IU/L).
- Prothrombin time: 24s (control: 12s)

Q: What is the likely diagnosis?

A: Obstructive jaundice

Q: Mention three important points in the history of the patient.

A: As follows:

- Generalized itching.
- Stool: Clay, pale or muddy coloured.
- Pain in the epigastrium or upper abdomen, anorexia and weight loss.

Q: Mention one investigation helpful for the diagnosis?

- A:**
1. Ultrasonography (USF) of whole abdomen (or hepatobiliary system)
 2. CT scan of abdomen
 3. ERCP
 4. MRCP

Q: Write down four important causes of this condition.

A: As follows:

- Choledocholithiasis
- Carcinoma of the head of pancreas.
- Periapillary carcinoma.
- Cholangiocarcinoma.

D/D of acute (days to weeks) painful jaundice :-

- Acute viral hepatitis
- Acute cholangitis
- Acute bidd-chiarri syndrome

D/D of chronic (weeks to months) painful jaundice :-

- HCC
- Mass in porta-hepatis
- Metastatic liver disease

D/D of chronic painless jaundice :-

- CA ampulla of vator
- CA head of pancreas

Case 02:-

A 26-year-old man presents with anorexia, nausea and repeated vomiting for 3 days. He also noticed high-coloured urine and pain in right upper abdomen for 2 days. investigation reveals:-

- Hb 14 gm/dL, WBCs 3900/cmm, polymorphs 46% lymphocytes 50%, eosinophil 4%.
- Serum bilirubin: 7.2 mg/dL (normal up to 1.2 mg/dL).
- SGPT: 800 IU/L (normal <20 IU/L).
- Serum alkaline phosphatase: 95 IU/L (normal 20-100 IU/L).

Q: Mention two physical signs you should look for.

A: As follows:

- Jaundice
- Liver is enlarged (soft in consistency , tender and smooth surface)

Q: What is the likely diagnosis?

A: Acute viral hepatitis.

Q: Mention one investigation that is helpful to see the prognosis.

A: Prothrombin time.

Q: Mention two other investigations.

A: As follows:

- Viral screening (HBsAg, anti-HAV, anti-HEV).
- Ultrasonogram of hepatobiliary system.

Q: Mention two other differential diagnoses.

A: As follows:

- Drug-induced hepatitis.
- Leptospirosis. (fever + hepato-renal involvement)
- Malaria (fever + hepato-renal involvement)

Q: Mention two complications.

A: As follows:

- Acute fulminating hepatic failure.
- Chronic liver disease.

Q: Mention one serious complication.

A: Acute fulminating hepatic failure.

Key points :-

With the above history and high bilirubin, high SGPT is suggestive of acute viral hepatitis. Alkaline phosphatase is normal or slightly high. Usually B and C infection may cause chronic liver disease. Hepatitis E may be serious, if associated with pregnancy.

Case 03:-

A 20 year old man presented with high coloured urine and yellow discolouration of the whole body. There is no history of anorexia, nausea or vomiting.

Laboratory investigations reveal:-

- Hb8gm/dL, WBCs 6600/cmm, polymorphs 65% lymphocytes 20%, platelet count 1,8000/cmm, ESR 95 mmHg in the 1st hour.
- Serum bilirubin: 3.2 mg/dL (normal up to 1.2 mg/dL).
- SGPT: 45IU/L (normal <20 IU/L).
- Serum alkaline phosphatase: 250 IU/L (normal 20-100 IU/L).

Q: What is the likely diagnosis?

A: Haemolytic jaundice.

Q: Mention three important physical finding in this patient.

A: As follows:

- Anaemia.
- Jaundice.
- Splenomegaly.

Q: Mention two hematological investigations to find out the cause.

A: As follows:

- Peripheral blood film (Shows microcytic hypochromic anaemia in hereditary haemolytic anaemia, macrocytic in autoimmune anaemia).
- Reticulocyte count (by supravital stain). High.

Q: Mention two further investigations to confirm your diagnosis:

A: As follows:

- Haemoglobin electrophoresis (to see type of hereditary haemolytic anaemia).
- Coombs test (to exclude autoimmune haemolytic anaemia).

Key points :-

Anaemia with high bilirubin but normal hepatic enzyme (normal SGPT, alkaline phosphatase) is highly suggestive of haemolytic anaemia. Mostly it may be due to hereditary haemolytic anaemia (e.g. thalassaemia major) or autoimmune haemolytic anaemia.

D/D of jaundice with anemia :-

- Hemolytic anemia (hereditary, autoimmune).
- CLD with portal hypertension.
- Primary liver malignancy (HCC)
- Metastatic liver disease (biliary or pancreatic neoplasm)

D/D Jaundice with splenomegaly :-

- CLD with portal hypertension
- Hemolytic anemia

D/D Jaundice with ascites :-

- CLD with portal hypertension
- Primary liver malignancy (HCC)
- Metastatic liver disease

D/D Jaundice with palpable gall bladder :-

- CA head of pancreas

D/D Jaundice with normal liver enzymes :-

- Non-hemolytic hereditary hyperbilirubinemia
- Hemolytic anemia

Case 04:-

A young student presented with weakness, anorexia, nausea and high coloured urine. He was suffering from sore throat 10 days back. There is history of previous similar attack. On examination: mild Anaemia, mild jaundice and liver is just palpable, nontender. Investigations show:

- CBC: Hb 10.2 g/dL, ESR 32 mm in 1st hour, WBCs 9900/cmm, polymorphs 61% lymphocytes 35%, monocytes 4%
- Reticulocyte count: 0.7% (normal 0.2 – 2%).
- Total bilirubin: 52 μ mol/L (normal <17 μ mol/L).
- Alanine transaminase (ALT) (SGPT): 15 IU/L. (normal <20 IU/L).
- USG of abdomen: Mild hepatomegaly.

Q: What is the likely diagnosis?

A: Gilbert syndrome.

Q: Suggest two investigations.

A: As follows:

- 48 h, 400 Kcal restriction test.
- IV 50 mg nicotine (there is rise of bilirubin).

Q: What treatment should be given in this patient?

A: As follows:

- Reassurance.
- Prolong fasting should be avoided.
- Therapeutic trial with phenobarbitone 60 mg TDS.

Key points :-

- Recurrent jaundice but presence of normal liver enzymes in a young patient is highly suggestive of Gilbert syndrome. It is a type of unconjugated nonhaemolytic hyperbilirubinaemia, occurs in 2-7% of normal individual, some cases inherited as autosomal dominant. Most patient remain asymptomatic. Jaundice is usually mild and occurs intermittently during infection or prolonged fasting. It is due to defect in the uptake of bilirubin by the liver and also there is a deficiency of UDP-glucuronyltransferase activity, which conjugates bilirubin with glucuronic acid. Liver biopsy is normal. Other cause of nonhaemolytic hyperbilirubinaemia are Crigler-Najjar syndrome (type 1 and 2), Dubin-Johnson syndrome (liver is black due to increased deposition of lipofuscin and melanin) and Rotor syndrome.
- Gilbert and Crigler-Najjar syndromes (unconjugated hyperbilirubinaemia)
 - Dubin-Johnson and Rotor syndromes (conjugated hyperbilirubinaemia)

Case 05:-

A 12 year old boy presents with scanty micturition and puffy face. Urine examination shows:

- Colour: Smoky.
- Pus cell 0-2/HPF.
- RBC 20-30/HPF.
- RBC cast: Present
- Prothrombin time: 24s (control: 12s)

Q: What is the probable diagnosis?

A: Acute glomerulonephritis.

Q: Mention one history you should take.

A: History of sore throat 1-3 weeks back.

Q: Write down three clinical signs you should look for.

A: As follows:

- Blood urea and serum creatinine.
- Serum electrolytes.
- Ultrasound of renal system.
- X-ray chest (PA view)

Q: Mention two complications.

A: As follows:

- Acute LVF.
- Acute renal failure.

Q: Write down principles of management.

A: As follows:

- Salt and fluid restriction.
- Diuretic such as furosemide.
- Antibiotic: Penicillin.

Key Points :-

History of sore throat followed by scanty, smoky urine associated with and periorbital puffiness is highly suggestive of acute glomerulonephritis. (usually post streptococcal glomerulonephritis).

Case 06:-

A 7 year old girl presented with swelling of the whole body with scanty micturition for 10 days.

Her urine examination reveals:

- Colour: Straw.
- Albumin +++
- RBC: Nil.
- Pus cell 0-2/high powered field (HPF).
- Fatty cast present.
- Prothrombin time: 24s (control: 12s)/

Q: What is the likely diagnosis?

A: Nephrotic syndrome.

Q: Mention three investigations.

A: As follows:

- Serum total protein and albumin.
- 24 urinary protein (>3 g).
- Serum lipid profile (there is high cholesterol and triglyceride).

Q: Mention one drug you think that should be given to this patient.

A: Prednisolone.

Key Points:-

Generalized oedema, massive proteinuria and hypoalbuminaemia are suggestive of nephrotic syndrome. Commonest cause in children is minimal change disease and in adult membranous or proliferative glomerulonephritis. Prognosis is better in children, but there may be relapse.

D/D of generalized body swelling with SOB :-

- CCF
- Constrictive pericarditis
- Pericardial effusion
- CKD with volume overload

D/D of generalized body swelling without SOB :-

- CLD
- CKD
- Nephrotic syndrome
- Malabsorption

Case 07:-

A 40 year old male presents with low grade fever, weakness, loss of appetite and difficulty in micturition. Urine examination shows:

- Albumin +.
- BRC 5-6/HPF.
- Pus cell 50-60/HPF.
- Culture: No growth.

Q: What does the urine report shows?

A: Sterile pyuria.

Q: Mention three causes.

A: As follows:

- Renal tuberculosis.
- Partially treated urinary tract infection.
- Analgesic nephropathy.
- Tubulointerstitial nephritis.

Q: Mention two commonest causes.

A: As follows:

- Renal tuberculosis.
- Partially treated urinary tract infection.

Q: Mention four further investigations.

A: As follows:

- Urine for acid fast bacteria (AFB) and mycobacterial culture.
- Ultrasound of renal system.
- Tuberculin test.
- Chest X-Ray.

Key Points:-

Presence of pus cells in the urine but sterile on culture is called sterile pyuria.
Tuberculosis of renal system is the likely cause.

Q:

A:

Q:

A:

Key P

Case 08:-

A 30 year old female presents with high grade temperature, anorexia, nausea, vomiting frequency of micturition and pain in the lumbar region. Urine examination shows:

- Albumin +.
- RBC few.
- Pus cell plenty.
- Culture: No growth.

Q: What is the likely diagnosis?

A: Acute pyelonephritis.

Q: Mention one investigation to confirm the diagnosis.

A: Urine culture and sensitivity.

Key Points:-

Any patient with frequency, burning or difficulty in micturition associated with high temperature is suggestive of acute pyelonephritis. Treatment should be given according to culture and sensitivity of urine.

Case 9:-

A 60 year old male presented with fever, headache, dry cough, frequency of micturition and weakness for 10 days. Cefixime was given by a general practitioner. After 5 days, the patient complains of scanty urine, puffiness of face, swelling of legs, polyarthralgia and multiple skin rashes. Investigations reveal:

- Full blood count: Hb 10.9 g/dL, WBCs 11,200/- cmm, polymorphs 64%, lymphocytes 28%, eosinophils 8%, ESR 110 mm in 1st hour, platelets 1,75,000/-/cmm.
- Urine: Pus cells – plenty, proteinuria (++) ; total protein 1 g/24h.
- Urea: 19 mmol/L (normal 2.5-6.6 normal/L).
- Creatinine: 833 μ mol/L (normal 60-120 mmol/L, chloride 90 mmol/L, potassium 6.1 mmol/L, bicarbonate 17 mmol/L.

Q: What is your diagnosis?

A: Acute interstitial nephritis leading to acute kidney injury (AKI) due to cefixime.

Q: Suggest two further investigations.

A: USG of kidney, renal biopsy.

Q: How to manage this case?

A: As follows:

- Offending drug should be stopped.
- Prednisolone.
- Dialysis for AKI

Key Points:-

This patient presents with fever, skin rash, eosinophilia and renal impairment shortly after antibiotic therapy. These findings are consistent with acute interstitial nephritis. It is hypersensitivity reaction to tubular interstitium.

Case 10:-

A 25 year old male presented with fever for 3 weeks and disorientation for 3 days. Cerebrospinal fluid (CSF) study shows:

- Pressure: High
- Colour: Clear
- Cytology: Total WBC 350/cmm, neutrophil 5%, lymphocyte 95%, no RBC.
- Biochemistry: Protein 300 mg/dL. (normal up to 40 mg/dL), sugar 50 mg/dL (low).
- Microbiology: No organisms were found in Gram stains.

Q: What is the likely diagnosis?

A: Tubercular meningitis.

Q: Mention four differential diagnoses.

A: As follows:

- Viral meningitis.
- Fungal meningitis.
- Sarcoidosis.

Q: Mention one other investigation in CSF that is helpful for your diagnosis.

A: ADA (adenosine deaminase).

Q: What is the other finding in CSF?

A: If it is kept overnight, there is cob web appearance.

Q: Write down two important physical signs.

A: As follows:

- Brudzinkski Neck sign.
- Kernig sign.

Q: What four other investigations will you suggest?

A: As follows:

- Complete blood count (CBC) with erythrocytic sedimentation rate (ESR).
- Tuberculin test.
- Chest X-ray (PA view).
- CT scan and MRI of head.

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Q: What is the finding on fundoscopy?

A: Choroid tubercle in retina.

Key Points:-

History of low grade fever, weight loss and anorexia, along with signs of meningitis and typical findings in CSF (high lymphocytes, high protein and low sugar) is highly suggestive of tuberculosis meningitis. It should be treated with standard antitubercular therapy for at least 9 months plus prednisolone 60 mg for 3 weeks and then should taper.

Complications:-

- cranial nerve palsies (false localizing sign)
- hydrocephalus
- syndrome of inappropriate antidiuretic hormone secretion (SIADH)
- seizure
- focal neurological deficit

Case 11:-

A 19 year old man presented with high grade fever for 2 days and disorientation for 4 h. neck rigidity and kerning sign are present. CSF examination shows:

- Colour: Turbid.
- Pressure: Increased.
- Biochemistry: Protein 190 mg/dL, sugar 16 mg/dL, chloride 670 mg/dL.
- Cytology: Total cells 6200 /cmm, polymorphs 96%, lymphocytes 4%.

Q: What is the likely diagnosis?

A: Pyogenic meningitis.

Q: What are the causes of bacterial meningitis?

A: As follows:

- **Neonate:** Gram negative bacilli (Escherichia coli, Proteus), Group B streptococci, Listeria monocytogenes,
- **Preschool child:** Haemophilus influenzae Neisseria meningitidis, Streptococcus pneumoniae, Mycobacterium tuberculosis.
- **Older child and adult:** Neisseria meningitidis, S. pneumoniae, L. monocytogenes, M. tuberculosis, staphylococcus aureus (skull fracture), H. influenza.

Q: What is the commonest organism?

A: N. meningitidis (also called meningococcal.)

Q: What are the complications?

A: As follows:

- Brain abscess.
- Hydrocephalus.
- Cranial nerve palsies.

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Q: What other physical findings will you look in the body?

A: Skin rash such as petechial or purpuric, which indicates meningococcal septicaemia.

Q: If the patient develops shock, what is the likely cause?

A: Bilateral adrenal haemorrhage (Waterhouse Friderichsen syndrome).

Q: What would you look for before doing lumbar puncture? Why?

A: I will do fundoscopy to see papilloedema (indicates raised intracranial pressure). If it is present, then lumbar puncture should be avoided because there may be herniation of cerebellar tonsils through foramen magnum, which compresses the vital centre in medulla oblongata and causes sudden death.

Key Points:-

In any patient with high fever, headache, nausea, vomiting, photophobia with signs of meningism, indicates pyogenic meningitis.

Case 12:-

A 24 year old man presented with fever, headache, nausea, vomiting for 5 days. Neck rigidity and Kernig sign are present. CSF examination shows:

- Colour: Clear.
- Pressure: Increased.
- Biochemistry: Protein 60 mg/dL, sugar 50 mg/dL, chloride 670 mg/dL.
- Cytology: Total cells 100 /cmm, polymorphs 2%, lymphocytes 98%.

Q: What is the likely diagnosis?

A: Acute viral meningitis.

Q: What are the common organisms?

A: Enteroviruses like Echovirus and coxsackie are the most common organism.

Key Points :-

Low-grade fever; headache; nausea; vomiting and CSF findings – clear, slightly high protein with normal glucose and chloride, with high lymphocyte is suggestive of viral encephalitis. There are usually no serious sequelae unless encephalitis is present.

Case 13:-

A 45 year old man presented with sudden, severe occipital headache followed by unconsciousness. Neck rigidity and Kernig sign are present. CSF shows:

- Colour: Red (Frank blood).
- Pressure: Increased.
- Biochemistry: Protein 100 mg/dL, sugar 60 mg/dL.
- Cytology: Plenty of RBCs, polymorphs 60% lymphocytes 40%.

Q: What is the likely diagnosis?

A: Subarachnoid hemorrhage.

Q: Mention two causes?

A: As follows:

- Rupture of berry aneurysm.
- Rupture of arteriovenous malformation.

Q: Mention one fundocopy finding.

A: Subhyloid hemorrhage.

Q: Suggest one investigation.

A: CT scan of head.

Q: Mention two other investigations.

A: As follows:

- Magnetic resonance angiography (MRA).
- Digital subtraction angiogram (DSA).
- Cerebral angiography.

Q: Mention two causes of haemorrhagic CSF.

A: As follows:

- Subarachnoid haemorrhage.
- Trauma.
- Bleeding disorders like haemophilia, Christmas disease.

Key Points:-

Sudden, severe thunderclap headache followed by unconsciousness and meningism is highly suggestive of subarachnoid haemorrhage. Control of hypertension, administration of nimodipine and surgical obliteration of the aneurysm by clipping is the mainstay of the treatment.

Case 14:-

A 35 year old lady presented with following blood report:

1. Hb 6 g/dL, ESR 70 mm in 1st hour.

2. WBC: 50 x 10⁹ / cmm.

- Neutrophil: 35%
- Lymphocyte: 28%
- Monocyte: 03%
- Eosinophil: 05%
- Atypical cell: 29%

3. Platelet: 100x10⁹ / cmm.

Q: What are the abnormalities in this blood picture?

A: Anaemia, thrombocytopenia, leukocytosis, high ESR, atypical cell,

Q: What is the likely diagnosis?

A: Acute leukaemia.

Q: Mention one further investigation.

A: Bone marrow study.

Key Points :-

Presence of atypical cell with high WBC count is suggestive of acute leukaemia, which may be either myeloid or lymphoid (differentiated by immunophenotyping through conventional PCR or FISH technique)

Case 15:-

A 45 year old man presented with weakness, loss of appetite and heaviness in the abdomen for 3 months.

Laboratory investigation reveals:

- Hb 9 g/dL, WBCs 1,50,000/cmm, polymorphs 45%, lymphocytes 7%, myelocytes 22%, metamyelocytes 14%, myeloblasts 3%, basophils 8%, platelets 2,10,000/cmm, ESR 90 mm in 1st hour.
- PBF: Normocytic normochromic anaemia.

Q: What are the abnormalities in this blood picture?

A: Anaemia, leukocytosis with myelocyte and meta-myelocyte, few blasts, high basophil and high ESR.

Q: What is your diagnosis?

A: Chronic myeloid leukaemia.

Q: What is the cause of abdominal discomfort?

A: Splenomegaly, which may be huge.

Q: What investigation do you suggest?

A: As follows:

- Bone marrow study.
- Cytogenetic analysis for Philadelphia chromosome.
- RNA analysis to see the presence of BCR – ABL gene product.
- Leukocyte alkaline phosphatase (LAP) score (decreases).

Q: What are the therapies that may cure this condition?

A: imatinib, α interferon and Bone marrow transplantation.

Key points:-

In a middle aged patient with huge splenomegaly and presence of anaemia, leukocytosis with myelocyte and metamyelocyte, few blasts, high basophil, high ESR is suggestive of chronic myeloid leukaemia (CML). Blastic crisis, myelofibrosis may occur, CML, may be associated with low LAP score, high uric acid, high vitamin B₁₂ and high LDH.

Phases of CML:-

- CML in chronic phase
- CML in accelerated phase
- CML in blast phase (crisis)

Case 16:-

A 65 year old man presented with generalized body ache for the last 3 months. Laboratory investigation reveals:

- Hb8 g/dL, WBC normal, platelet 2,00,000/cmm.
- ESR: 115 mm in 1st hour.
- PBF: Normocytic normochromic anaemia with increased rouleaux formation.
- Blood Ca^{++} . High.

Q: What is the likely diagnosis?

A: Multiple myeloma.

Q: Write down two important investigations to confirm the diagnosis?

A: As follows:

- Bone marrow study (shows atypical plasma cells).
- 24 hour urine immunofixation test for (bence jones protein)
- Serum protein electrophoresis or immune electrophoresis.
- X-ray skull (shows multiplex lytic lesions).

Q: Write down three complications.

A: As follows:

- Pathological fracture of bone.
- Repeated infection.
- Bone marrow failure.
- Renal failure.
- Hyperviscosity syndrome.

Q: Mention one drug that may be curative.

A: Bortezomib.

Key Points :-

Elderly patient may present with generalized bodyache, unexplained anaemia, repeated infection, spontaneous fracture, hyperviscosity syndrome, bleeding disorder, renal failure is suggestive of multiple myeloma. Blood examination shows very high ESR with marked rouleaux formation in peripheral blood film (PBF). Bone marrow study shows presence of atypical plasma cells (usually >20%). Urine shows Bence-Jones proteinuria.

Case 17:-

A 40 year old male presented with generalized weakness and gum bleeding for 2 months. Investigation reveals:

- Hb: 7 g/dL.
- WBC: 15,000/mm³.
- Neutrophil: 16%
- Lymphocyte: 81%
- Platelet: 20,000/mm³.
- ESR: 70 mm in 1st hour.

Q: What is the hematological diagnosis?

A: Pancytopenia.

Q: Differential diagnosis of bicytopenia / pancytopenia .

A: primary bone marrow causes :-

- Leukemia
- Aplastic anemia
- Multiple myeloma

Non-primary bone marrow causes :-

- Infections (hepatitis B, HIV and brucellosis)
- CTDS (SLE)
- Nutritional deficiency (vitamine B12 deficiency)
- Hypersplenism
- Cytotoxic drugs / toxins / herbel medications

Q: Mention one investigation to confirm the diagnosis?

A: Bone marrow study.

Key points:-

Anaemia, leucopaenia and thrombocytopaenia indicates pancytopenia. Commonest cause is aplastic anaemia.

Case 18:-

A 29 year old female presented with weakness, palpitation and shortness of breath on exertion. Laboratory investigation reveals:

- Hb: 8.5 mg/dL.
- RBC, WBC and platelet counts are within normal limits.
- PBF shows hypochromic.
- MCV 65 fL (normal >80 fL).

Q: What is hematological diagnosis?

A: Hypochromic microcytic anaemia.

Q: Mention four causes.

A: As follows:

- Iron deficiency anaemia.
- β -Thalassaemia.
- Sideroblastic anaemia.
- Anaemia of chronic disorder.

Q: Which one is more likely diagnosis in this patient?

A: Iron deficiency anaemia.

Q: What is the commonest cause of iron deficiency anaemia?

A: Bleeding (menorrhagia, hemorrhoid).

Q: What history do you like to take in this female patient?

A: History of menorrhagia.

Q: What further investigation will you do?

A: As follows:

- Iron profile (serum iron, serum ferritin, total iron binding capacity (TIBC))
- Hemoglobin electrophoresis.
- Bone marrow study.

Key Points:-

Microcytic hypochromic blood picture with history of blood loss suggests iron-deficiency anaemia. Serum iron and ferritin will be low and TIBC will be high. Treatment includes correction of anaemia by blood transfusion, iron therapy for 3-6 months (to replenish the store) and treatment of primary cause.

Causes of iron deficiency anemia :-

- Malnutrition
- Blood loss (GI blood loss and menstruation)
- Decreased absorption (autoimmune gastritis , H-pylori gastritis and celiac sprue)
- Increased requirement (pregnancy and lactation)

Treatment of iron deficiency anemia :-

- Oral (ferrous sulfate 325mg once daily)
- Total duration of therapy is 6 months
- HCT is halfway toward normal within 3 weeks and full returns to baseline after 2 months .
- Parenteral iron therapy.

Indications for parenteral iron therapy :-

- Intolerance or refractoriness (define as haemoglobin increment of less than 1g/dl after 4 to 6 weeks of 100mg per day of elemental oral iron)
- GI disease (IBD)
- Chronic hemodialysis

Case 19:-

A 15 year old girl presented with weakness, abdominal distension and loss of appetite. Examination of the abdomen reveals huge splenomegaly and mild hepatomegaly. Laboratory results show:

- Hb 7%, WBC 7200/cmm, neutrophil 62% lymphocyte 23% =, platelet 2,30,000/cmm.
- Mean cell volume (MCV): 69 fL (normal >80).
- Serum bilirubin: 3.1 mg/dL.

Q: What is the likely diagnosis?

A: β -Thalassemia major.

Q: What is the triad of signs in this condition?

A: As follows:

- Anaemia.
- Jaundice.
- Splenomegaly

Q: What history is important in such condition?

A: Family history of such illness.

Q: What physical sign will you look for?

A: Anaemia, jaundice, splenomegaly, frontal and parietal bossing, mongoloid faces.

Q: What investigation will you do to confirm the diagnosis?

A: Haemoglobin electrophoresis.

Q: How to treat?

A: **1. General treatment:**

- Blood transfusion to keep the haemoglobin above 10 g/dL., usually every 4 months (life span of RBC is 4 months)
- Folic acid daily (1mg per kg)
- Iron containing drugs (sulfonamide) and diet should be avoided.
- Desferrioxamine to prevent transfusion haemosiderosis.
- Erythropoietin and hydroxyurea may be given.

2. Specific treatment:

- Allogenic bone marrow transplantation,
- If huge splenomegaly with features of hypersplenism (increase transfusion requirement or refractory symptoms) **Splenectomy** may be done.

Key Points :-

Presence of microcytic hypochromic anaemia with high reticulocyte count, jaundice and splenomegaly in a young patient indicates hereditary haemolytic anaemia, most commonly β -Thalassemia major.

Case 20:-

A 50 years old woman presented with weakness and anorexia for 3 months. Her laboratory investigations reveal:

- Hb 8% WBC 6700/cmm, neutrophil 65%, lymphocytes 25%, platelet 2,10,000/cmm.
- Reticulocyte: 1%
- MCV: 112 fL. (normal up to 96 fL).
- PBF shows macrocytosis and hypersegmented neutrophils.
- Serum bilirubin: 1.0 mg/dL.

Q: What is the hematological diagnosis?

A: Macrocytic anaemia.

Q: What are the causes of macrocytosis?

A: As follows:

- Megaloblastic anaemia (due to B₁₂ or folic acid deficiency)
- Hypothyroidism.
- Chronic liver disease.
- Chronic alcoholism.
- Hemolysis.
- Azathioprine therapy.

Q: What next investigation should be done?

A: Bone marrow study.

Q: What are the findings in bone marrow study and how to interpret?

A: As follows:

- If macrocytosis is associated with megaloblast in bone marrow: The diagnosis is megaloblastic anaemia.
- If macrocytosis is associated with normoblastic bone marrow: The causes are hypothyroidism, chronic liver disease, chronic alcoholism, haemolysis, azathioprine therapy,

Q: What other investigations do you suggest?

A: Serum vitamin B₁₂ and folic acid assay.

Key Points:-

Anaemia with high MCV indicates macrocytic anemia, Most common cause is megaloblastic anaemia due to vitamin B₁₂ or folic acid deficiency.

1. Causes of vitamin B 12 deficiency :-

- Dietary deficiency
- Decreased production or absorption of intrinsic factor (pernicious anemia , gastrectomy , H-pylori infection)

2. Decreased ileal absorption of vitamin B12 :-

- Surgical resection
- Crohn's disease

3. Competition of B12 in the gut :-

- Blind loop syndrome
- Fish tapeworm

4. Pancreatic insufficiency and use of proton pump inhibitors**Management of vitamin B12 deficiency :-**

- Intramuscular or subcutaneous injection of 100 to 1000 mcg of vitamin B₁₂ is given daily for first week, weekly for month then monthly for whole life .
- Hypokalemia may complicate the first several days of therapy
- A brisk reticulocytosis occur in 5 to 7 days and hematological picture normalizes in 2 months .
- CNS symptoms and signs are potentially reversible if they have been present for less than 6 months .

Case 21:-

A 60 year old man presents with fever, cough and weight loss. She was suffering from breast cancer, which was treated by mastectomy and chemotherapy. Her blood picture shows:

- Hb 11.4 g/dL, WBCs 23,700/cmm, polymorphs 82%, lymphocytes 6%, metamyelocytes 3%, myelocytes 3%, promyelocytes 1%, myeloblast 1%, erythroblast 4%, platelets 1,80,000/cmm.
- ESR: 10 mm in 1s hour.
- Chest X-ray: Absent left breast shadow.

Q: What is the hematological diagnosis?

A: Leukemoid reaction.

Q: What is the likely cause?

A: Bone marrow infiltration from carcinoma of breast.

Q: Mention one further investigation to confirm your diagnosis?

A: Bone marrow study.

Key Points:-

Leukemoid reaction means that the peripheral blood picture resembles leukaemia; but there is no leukaemia.

Causes of myeloid leukemoid reaction:-

- Infections
- Malignancy

Causes of lymphoid leukemoid reaction:-

- Infections (TB, CMV and infectious mononucleosis)
- Malignancy

Case 22:-

A 65 year old woman presented with pain in the epigastrium, loss of appetite, fever, generalized bodyache, night sweating, occasional headache, dizziness and weight loss for 3 months. She is pale and emaciated. There is massive splenomegaly.

Investigations reveal:

- Hb9.1 g/dL, WBC 18,500/cmm, polymorphs 60%, lymphocytes 28%, myelocytes 6%, metamyelocytes 2%, myeloblasts 1%, erythroblast 3%, ESR 93 mm in 1st hour, platelets 6,65,00 / cmm.
- Liver function tests: Serum bilirubin 28 μ mol/L, SGPT 38 IU/L, alkaline phosphatase 200 IU/L.
- Chest X-ray: Normal.

Q: What is the hematological diagnosis?

A: Leukoerythroblastic anaemia.

Q: What is the likely diagnosis?

A: Myelofibrosis.

Q: What additional findings may be seen in blood film?

A: Tear-drop poikilocytes.

Q: What are the other causes of such blood picture?

A: As follows:

- Lymphoma.
- Multiple myeloma.
- Secondary deposits in bone marrow
- Active hemolytic anaemia.

Q: Suggest two other tests.

A: As follows:

- Bone marrow study: May be dry tap. Trephine biopsy is needed, which shows increased megakaryocyte, increased reticulum and fibrous tissue.
- LAP score: High.

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Q: Mention one dangerous complication.

A: Transform to acute myeloid leukaemia. (AML) (10-20% cases).

Q: What treatment should be given?

A: As follows:

- Blood transfusion.
- Folic acid.
- Hydroxycarbamide (hydroxyurea) may be given.
- In young patient, bone marrow transplantation.
- Radiotherapy for huge spleen.
- If evidence of hypersplenism and huge spleen with pressure symptoms, splenectomy may be necessary.

Key Points:-

In any elderly patient with huge splenomegaly and leukoerythroblastic blood picture, the likely cause is myelofibrosis. PBF shows tear-drop. Myelofibrosis is a disorder of unknown cause, characterized by bone marrow fibrosis, extramedullary haemopoiesis and leukoerythroblastic blood picture due to neoplastic proliferation of primitive stem cells. It is common above 50 years. There may be peptic ulcer, pruritus after hot bath, gout, etc.

Case 23:-

A 53 year old man is diagnosed as a case of chronic granulocytic leukaemia. He was treated with chemotherapy and responded well. Three years later, the patient presented with the complaints of severe weakness, loss of weight, lethargy, epistaxis and pain in left upper abdomen.

Investigations show:

- Hb7.5 g/dL, WBC 12,800/cmm, platelets 1,00,000/cmm, RBC 2.8 million/cmm, ESR 85 mm in 1st hour.
- Differential count: Neutrophils 34%, basophil 8% lymphocyte 6%, myeloblast 40%, myelocyte 10%, metamyelocyte 2%.

Q: What is the diagnosis?

A: CML in Blastic crisis .

Q: What is the cause of abdominal pain?

A: Splenic infarction.

Q: Suggest one investigation to confirm your diagnosis.

A: Bone marrow study.

Key Points:-

Appearance of increased number of blast cells in PBF in a patient with CML suggests blastic crisis. It may be myeloid or lymphoid.

Case 24:-

9 days after recovering from a viral fever, a 13 year old girl presents with several small bruises on her arms and legs.

Investigations reveal:

- Full blood count: HB 11.3 g/dL, WBCs 10,700/cmm, polymorphs 59%, lymphocytes 38%, ESR 60 mm in 1st hour. Platelet count 80,000/cmm.
- Blood film: Normocytic and normochromic.
- Chest X-ray: Normal.

Q: What is the likely diagnosis?

A: Idiopathic thrombocytopenic purpura.

Q: Suggest two diagnostically useful investigations.

A: Bone marrow study (increase megakaryocytes) and antiplatelet antibody.

Q: What disease should be excluded in such finding?

- A:**
- Infections (dengue fever, malaria, hepatitis B and C, HIV)
 - Connective tissue diseases (SLE)

Treatment of I.T.P :-

- Patient with platelets count <25000 to 30000 or those with significant bleeding should be treated with corticosteroids with or without IVIG, other patients should be monitored periodically
- Response to corticosteroids generally seen in 3 to 7 days and response to IVIG generally seen in 24 to 36 hours
- Adding rituximab to corticosteroids has first line treatment maybe improved the initial response rate.
- Romiplostin or eltrombopag is second line therapy (when not responding to ITP-specific therapy)

Indications for BM biopsy :-

- Un-explained cytopenia in two or more lineage
- Patient with age >40 years with isolated thrombocytopenia

Indication for splenectomy:-

- Severe thrombocytopenia not responding to second line therapy

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Key Points:-

purpuric spot in a patient with recent history of viral fever, associated with low platelet count and increased megakaryocytes in bone marrow is suggestive of idiopathic thrombocytopenic purpura (ITP). In ITP, low platelet, increased megakaryocyte in bone marrow prolonged bleeding time, normal clotting time are common.

Case 25:-

A 41 year old man presented with frequent headache, dizziness, lack of concentration, pruritus and heaviness in the left upper abdomen for 3 months. Laboratory investigations reveal:

- Hb 17.6 g/dL, WBCs 21,000/cmm, polymorphs 83%, lymphocytes 15% eosinophils 2%, RBCs 8.2 million/cmm, platelets 8,50,000/cmm, ESR 1 mm in 1st hour.
- Packed cell volume (PCV): 65% (normal up to 45%)
- Random blood sugar (RBS): 7.2 mmol/L.
- Chest X-ray: Normal.

Q: What is your diagnosis?

A: Polycythemia rubra vera (PRV).

ESR ↓

Q: Suggest one investigation.

A: Measurement of red cell mass (increased). ✓

Q: Suggest one treatment.

A: Venesection.

Q: What drugs can be used?

A: Radioactive phosphorus, hydroxyurea, interferon α .

Q: Mention our complications.

A: As follows:

- Hypertension.
- AMI
- Thromboembolism (cerebral, coronary).
- Peptic ulcer.
- Gout
- Myelofibrosis.

Key Points:-

This patient has high haemoglobin and RBC, also high PCV, which suggests polycythemia. High WBC and platelet count associated with splenomegaly are all in favour of polycythemia rubra vera. Red cell volume is high in PRV. There is increased LAP (leukocyte alkaline phosphatase), vitamin B₁₂ and uric acid (may cause gout). Bone marrow shows hypercellular with increased megakaryocyte.

Case 26:-

A 29 year old woman was admitted in the hospital with the complaints of PV (blood loss per vaginum) bleeding and high grade, continuous fever, following a self-induced abortion. She looks toxic anaemia severe, BP 80/60 mmHg, pulse 124/min, temperature 39.6°C. Lower abdomen very tender.

Investigations reveal:

- Full blood count: Hb 5.0 g/dL, WBCs 28,700/cmm, polymorphs 87%, lymphocytes 13%, platelets 30,000/cmm, ESR 85 mm in 1st hour.
- PBR: Microcytic, hypochromic and fragmented RBC.
- Prothrombin time: 30s (control 12).
- APTT: 60 s (control 32).
- Fibrinogen: 0.05 g/L. (1.5 – 4.0 g).

Q: What is the main diagnosis in this blood disorder?

A: Disseminated intravascular coagulation (DIC).

Q: Mention four abnormalities in this blood picture.

A: DIC with microangiopathic hemolytic anaemia with iron deficiency anaemia along with septicemia.

Q: Give three possible causes of haematological abnormality.

A: Septicaemia, retained product of conception or amniotic fluid embolism.

Key Points:-

DIC is a hemorrhagic disorder in which diffuse intravascular clotting causes a haemostatic defect resulting from utilization of coagulation factor and platelet in the clotting process. There is secondary activation of fibrinolysis, leading to production of fibrin degradation products (FDP). The consequence of these changes is a mixture of initial thrombosis, followed by bleeding tendency due to consumption of coagulation factors and fibrinolytic activity.

Case 27:-

A 46 year old woman is admitted in the emergency department with shortness of breath, cough, swelling in both feet and extreme weakness. Respiratory function tests reveal:

- Vital capacity: 2.0 L (predicated 2.4—3.6 L).
- FEV1: FVC ratio 2.1 L (predicted 2.25-3.25 L).
- Transfer factor: 4.1 L (predicted 5.8-8.7 L).
- Residual volume: 1.1 L (predicted 1.55-2.32 L).

Q: What pulmonary defect is present?

A: Restrictive lung disease.

Q: What is the likely diagnosis with the above findings?

A: Interstitial lung disease.

Q: Suggest five investigations to find out cause.

A: As follows:

- Chest X-ray.
- CT scan of chest
- ABG (arterial blood gas) analysis.
- Bronchoscopy and bronchoalveolar lavage.
- Transbronchial or open lung biopsy.

Q: What are the causes of restrictive lung disease?

A: As follows:

- Sarcoidosis.
- DPLD
- Ankylosing spondylitis.

Key Points:-

In restrictive lung disease, both FVC and FEV are proportionately reduced; but ratio of this two is normal. Total lung capacity is reduced and residual volume is reduced or normal. CO transfer factor is also low.

Q: classify DPLD:-

DPLD is classified into six groups:-

1. Granulomatous DPLD (e.g. sarcoidosis).
2. Granulomatous DPLD with vasculitis (e.g. Wegener granulomatous, Churg-Strauss syndrome, microscopic vasculitis).
3. Idiopathic interstitial pneumonia (IIP):
 - a. Idiopathic pulmonary fibrosis [IPF, also called previously called cryptogenic fibrosing alveolitis 90%].
 - b. Idiopathic interstitial pneumonia other than IPF (10%)
 - Desquamated pneumonia.
 - Acute interstitial pneumonia.
 - Nonspecific interstitial pneumonia.
 - Respiratory bronchiolitis.
 - Cryptogenic organizing pneumonia [(COP, also called bronchiolitis obliterans organizing pneumonia (BOOP))].
 - Lymphocytic interstitial pneumonia.
4. Pulmonary autoimmune rheumatic diseases (e.g. rheumatoid arthritis, SLE).
5. Drugs (busulfan, bleomycin, methotrexate, nitrofurantoin, amiodarone).

Case 28:-

A lady of 42 years, housewife, has been suffering from breathlessness, occasional dry cough and weight loss for 2 years.

Her lung function test shows:

- FVC: 2.00 L (predicted 2.4-3.6 L).
- FEV: 1.45 L (predicted 2.25-3.25 L).
- RV: 2.89 L (predicted 1.5-2.32 L).
- FRC: 3.56 L (predicted 2.17-3.25 L).
- TLC: 5.86 L (predicted 3.96-5.66 L).
- TLCO: 4.7 mmol/min/kPa (predicted 5.8-8.7 L).

Q: What is the lung function test?

A: Obstructive airway disease.

Q: What is the most likely diagnosis?

A: Emphysema.

Q: Suggest two possible causes.

A: α_1 -Antitrypsin deficiency, cigarette smoking.

Q: Name two obstructive airway diseases.

A: Chronic obstructive pulmonary disease (COPD), bronchial asthma.

Key Points:-

In obstructive lung disease, forced expiratory volume in 1 second (FEV) is markedly reduced; forced vital capacity (FVC) is also reduced. The ratio of these two is also reduced. CO transfer factor is low. Residual volume and total lung capacity are high.

Case 29:-

A 52 year old woman presented with frequency of micturition, extreme weakness, loss of appetite, constipation, abdominal pain and weight loss for 7 months. Investigation reveal:

- Full blood count: Hb 10.6 g/dL, WBCs 8,900/cmm, polymorphs 55%, lymphocytes 43%, monocytes 2% ESR 90 mm in 1st hour.
- Plain X-ray KUB (kidneys, ureters, bladder); Nephrocalcinosis
- USG of abdomen: Calcification in both kidneys.
- Urine: Protein (+), few pus cells.
- RBS: 6.9 mmol/L.
- Creatinine: 1.4 mg/dL.
- Serum electrolytes: Sodium 139 mmol/L. chloride 110 mmol/L, potassium 3.6 mmol/L, bicarbonate 22 mmol/L.

Q: What is the likely diagnosis?

A: Primary hyperparathyroidism.

Q: Suggest five investigations to confirm your diagnosis.

A: As follows:

- Serum calcium, phosphate and alkaline phosphatase.
- Parathyroid hormone assay.
- Hydrocortisone suppression test.
- X-ray of hand (to see sub periosteal erosion in the medial side of phalanges) and X-ray of skull (to see pepper-pot appearance).
- USG or CT scan of neck (parathyroid adenoma or hyperplasia).
- Other test: Thallium/technetium subtraction scan of thyroid and parathyroid.

Q: What single treatment should be given immediately?

A: Plenty of fluid (infusion of normal saline 4-6 L daily) for hypercalcaemia.

Key Points :-

The above symptoms associated with hypercalcaemia and nephrocalcinosis indicates hyperparathyroidism. It may be **primary** (due to adenoma, hyperplasia or carcinoma of parathyroid), **secondary** (chronic renal failure, malabsorption, like celiac disease, crohn's disease and rickets or osteomalacia) or tertiary (autonomous from). In mild and asymptomatic case follow-up. Total parathyroidectomy with transplantation of parathyroid in the forearm muscles is done in primary hyperparathyroidism. Surgery is also required for tertiary hyperparathyroidism. Treatment of secondary causes should be done.

Case 30:-

A 32 year old lady presented with bilateral popptosis, gritty sensation and discomfort in both eyes, palpitation and weight loss. Investigation reveals:

- Serum FT₃: 9.0 pmol/L (normal 1.3-3.5 pmol/L).
- Serum FT₄: 215 pmol/L (normal 70-160 pmol/L).
- TSH: 0.04 mIU/L (normal 0.5-5.1 mIU/L).

Q: What is your diagnosis?

A: Thyrotoxicosis due to Graves disease.

Q: Write down two other causes of such investigation findings?

A: As follows:

- Toxic multinodular goiter.
- Toxic adenoma.

Q: Write down two important finding by examining of this patient.

A: As follows:

- Tremor of the outstretched hand.
- Deep tendon Jerks are exaggerated
- Palms are warm and sweaty
- Sleeping pulse (tachycardia)

Q: Write down four other important investigations.

A: As follows:

- Radio-iodine uptake test.
- Thyroid scan.
- USG of neck.
- TSH receptor antibody (TRAb) test.

Q: Write down the modalities of treatment.

A: As follows:

- Ant thyroid drugs: Carbimazole (20 to 30 mg) or propylthiouracil. 200 to 300 mg
- Propranolol 60mg per day twice or thrice (maximum 300mg per day) .
- Radioactive iodine therapy.
- Sub total / total Thyroid surgery.

Key Points :-

Graves disease is characterized by diffused goiter, exophthalmos and dermopathy. The natural history of Graves disease is hyperthyroidism, followed by euthyroid and later hypothyroidism. TSH receptor antibody (TRAb) is high.

Case 31:-

A 35 year old female presented with generalized weakness and weight gain. Her thyroid function test reveals:

- Serum FT₃: 2pmol/L. (normal 1.3-3.5 pmol/L).
- Serum FT₄: 7pmol/L (normal 70-160 pmol/L).
- TSH: 35mIU/L (normal 0.5-5.1 mIU/L).

Q: What is the most likely clinical diagnosis?
A: Primary hypothyroidism.

Q: Mention four important clinical signs of this patient.
A: As follows:

- Hoarseness or croaky voice.
- Puffiness of face.
- Bradycardia
- Delayed relaxation of ankle jerk.
- Dry skin

Q: What are the ECG changes in hypothyroidism?
A: As follows:

- Sinus Bradycardia.
- Low-voltage ECG tracing.
- T-wave inversion.

Q: What is the treatment?
A:

Life long thyroxin (1.6 mcg/kg/day) replacement, and then gradually increasing the dose. According to clinical response and serum TSH measuring every 4 to 6 weeks.

Key Points :-

Weight gain, increased sleepiness, cold intolerance and constipation are the usual features of hypothyroidism.

Case 32:-

A 65 year old obese woman is hospitalized with the complains of fever, cough with purulent sputum for 3 days. Investigations reveal:

- Full blood count: Hb 10.5 g/dL, WBCs 18,540/cmm, polymorphs 82%, lymphocytes 16% monocytes 2%, platelets 1,80,000/cmm.
- Chest X-ray: Consolidation (left upper zone).
- ECG: Low-voltage tracing and sinus bradycardia.
- Serum FT₃: 2.2 pmol/L (normal 1.3-3.5 pmol/L).
- Serum FT₄: 104 pmol/L (normal 70-160 pmol/L).
- TSH: 0.2 mIU/L (normal 0.5-5.1 mIU/L).

Q: What is the likely diagnosis of thyroid disorder?

A: Sick euthyroid syndrome.

Q: What further investigation would you suggest?

A: Repeat FT₃, FT₄ and TSH after cure of pneumonia.

Q: What is the cause of ECG abnormality?

A: Obesity.

Key points:-

In any non-thyroidal illness [acute myocardial infarction, pneumonia, cardiovascular disease (CVD)], there may be abnormal thyroid function tests: although the patient is euthyroid. This is called sick euthyroid syndrome. There may be low total and free T₃ and T₄ along with low or normal thyroid stimulating hormone (TSH). It is confused with secondary hypothyroidism. The tests should be repeated after recovery from the systemic disease, usually after 6 weeks.

Appropriate use of thyroid tests:-

	Test	Comment
For screening	Serum thyroid-stimulating hormone (TSH) free thyroxine (FT ₄)	Most sensitive for primary hypothyroidism and hyperthyroidism excellent test
For hypothyroidism	Serum TSH antithyroperoxidase and antithyroglobulin antibodies	High in primary and low in secondary hypothyroidism elevated in hashimoto thyroiditis
For hyperthyroidism	Serum TSH triiodothyronine or (FT ₃) ¹²³ I uptake and scan antithyroperoxidase and antithyroglobulin antibodies, Thyroid-stimulating immunoglobulin (TSI)	Suppressed except in TSH-secreting pituitary tumor or pituitary hyperplasia (rare) elevated increased uptake; diffuse versus "hot" foci on scan elevated in graves disease usually (65%) positive in graves disease.
For thyroid nodules	Fine-needle aspiration (FNA) biopsy ¹²³ I uptake scan, ^{99m} Tc, scan ultrasonography	Best diagnostic method for thyroid cancer cancer is usually "cold"; less reliable than FNA biopsy vascular versus avascular Useful to assessing FNA biopsy. Useful in assessing the risk of malignancy (multinodular goiter or pure cysts are less likely to be malignant). Useful to monitor nodules and patients after thyroid surgery for carcinoma.

Data interpretation of thyroid function test :-

Diagnosis	TSH	Free T4	Notes
Thyrotoxicosis (e.g. Graves' disease)	Low	High	In T3 thyrotoxicosis the free T4 will be normal
Primary hypothyroidism (primary atrophic hypothyroidism)	High	Low	
Secondary hypothyroidism	Low	Low	Replacement steroid therapy is required prior to thyroxine.
Sick euthyroid syndrome (non-thyroidal illness)	Low	Low	Common in hospital inpatients T3 is particularly low in these patients, TSH may be normal in some cases rT3 is used to differentiate
Poor compliance thyroxine	High	Normal	The TSH level is high. This implies that over recent days/weeks her body is thyroxine deficient. However, the free T4 is within normal range. The most likely explanation is that the Pt. started taking the thyroxine properly just before the blood test. This would correct the thyroxine level T4, T4 is converted to T3. But the TSH takes longer to normalise. (N free T4, free T3, but $\uparrow$ TSH)
Sub-clinical hypothyroidism	High	Normal	

Case 33:-

A 32 year old woman presents with fever, dry cough, pain in the throat, headache, bodyache, palpitation and intolerance to heat for 1 week. There is fine tremor of the outstretched hands. Palms are warm and sweaty. Thyroid is diffusely enlarged, soft, tender, no bruit. Investigations reveal:

- Full blood count: Hb 11.1 g/dL, WBCs 3,500/cmm, polymorphs 48%, lymphocytes 52%, ESR 110 mm in 1st hour, platelets 3,65,000/cmm.
- Chest X-ray: Normal.
- Serum FT₃: 8.7pmol/L (normal 1.3-3.5 pmol/L).
- Serum FT₄: 205pmol/L (normal 70-160 pmol/L).
- TSH: 0.4mIU/L (normal 0.5-5.1 mIU/L).
- Radio-iodine uptake: After 2 h, 7% (normal 5-15%). After 24 h, 5.1% (normal 15-30%).

Q: What is the likely diagnosis?

A: De Quervain thyroiditis (sub-acute thyroiditis).

Q: Suggest one investigation.

A: Fine needle aspiration cytology (FNAC) of thyroid gland.

Q: Mention three other causes of such thyroid functions (high T₃, T₄ but low radio-iodine uptake)

A: As follows:

- Postpartum thyroiditis.
- Factitious hyperthyroidism.
- Iodine-induced hyperthyroidism.

Key Points :-

De Quervain thyroiditis is a virus-induced transient inflammation of thyroid gland, usually self-limiting. It is caused by Coxsackie B4 also may be associated with influenza, infectious mononucleosis, measles, mumps, common cold. It is more in females, 20-40 years. Presents with pain over thyroid, radiate to jaw, ear, and gets worse by coughing, swallowing or movement of neck. Systemic features are common. Thyroid gland is enlarged and tender. FNAC shows presence of giant cells. ESR is typically high. Inflammation of thyroid releases thyroid hormones, which are high in the blood, responsible for the features of thyrotoxicosis. This may persist for 4-6 weeks. Iodine uptake is low. Hyperthyroidism followed by transient hypothyroidism may occur. Complete recovery occurs in 4-6 months.

Treatment: Symptomatic (NSAID). Propranolol may be used. Prednisolone 40 mg/day for 3-4 weeks may be needed. Antithyroid drug should not be given.

Case 34:-

A young lady with 4 months pregnancy presented with the complains of palpitation, excessive sweating and intolerance to heat. Thyroid gland is diffusely enlarged and nontender, soft.

Investigation reveals:

- Full blood count: Hb 10.5 g/dL, WBCs 9,800/cmm, polymorphs 66%, lymphocytes 30%, eosinophils 4%, platelets 2,00,000/cmm.
- Serum total T₃: 9pmol/L (normal 1.32-3.1pmol/L).
- Serum total T₄: 290pmol/L (normal 70-160 pmol/L).
- Serum TSH: 4 mIU/L (normal 1.52-5mIU/L).

Q: What is your inference from the above result?

A: Euthyroid state.

Q: What further investigations should be done?

A: FT₃ and FT₄

Q: Mention four causes of such thyroid function abnormality.

A: Pregnancy, oral contraceptive pill, oestrogen therapy, acute intermittent porphyria.

Key Points:-

Patient's symptoms are due to pregnancy. Thyroid gland may be normally enlarged in pregnancy, so her total T₃ and T₄ are high, but TSH is normal. Any causes of high thyroxine-binding globulin (TBG) will also cause high total T₃ and total T₄ but FT₃ and FT₄ will be normal. Alternately, any cause of low TBG will also cause low total T₃ and total T₄.

Causes of high TBG:- Congenital or hereditary, pregnancy, drug (oestrogen, clofibrate, phenothiazine), oral contraceptive pills, acute intermittent porphyria, acute viral hepatitis; also in hypothyroidism.

Causes of low TBG:- Hereditary, malnutrition, nephrotic syndrome, liver failure, drugs (sulphonylurea, salicylate, phenytoin, phenylbutazone, anabolic steroid, androgen, corticosteroid), active acromegaly. Also in hyperthyroidism.

Case 35:-

A housewife of 40 year, presented with frequent headache, vertigo and insomnia for 2 months. Blood pressure (BP) is 160/100 mmHg. Investigations reveals:

- Full blood count: Hb 11.5 g/dL, WBCs 8300/cmm, polymorphs 64%, lymphocytes 33%, monocytes 3%, ESR 48 mm in 1st hour, platelets 2,70,000/cmm.
- Urine: Glycosuria (++), proteinuria (+).
- Serum electrolytes: Sodium 145 mmol/L, chloride 101 mmol/L, potassium 3.1 mmol/L, bicarbonate 30 mmol/L.
- Creatinine: 1.3 mg/dL.
- Chest X-ray: Heart slightly enlarged.
- Fasting blood sugar (FBS): 9.0 mmol/L.

Q: Suggest three differential diagnoses.

A: Cushing syndrome, primary aldosteronism, ectopic adrenocorticotrophic hormone (ACTH) syndrome.

Q: What is the likely diagnosis in this case?

A: Cushing syndrome.

Q: What five physical signs will you see?

A: central Obesity, plethoric face, skin is thin with bruise and striae with proximal myopathy.

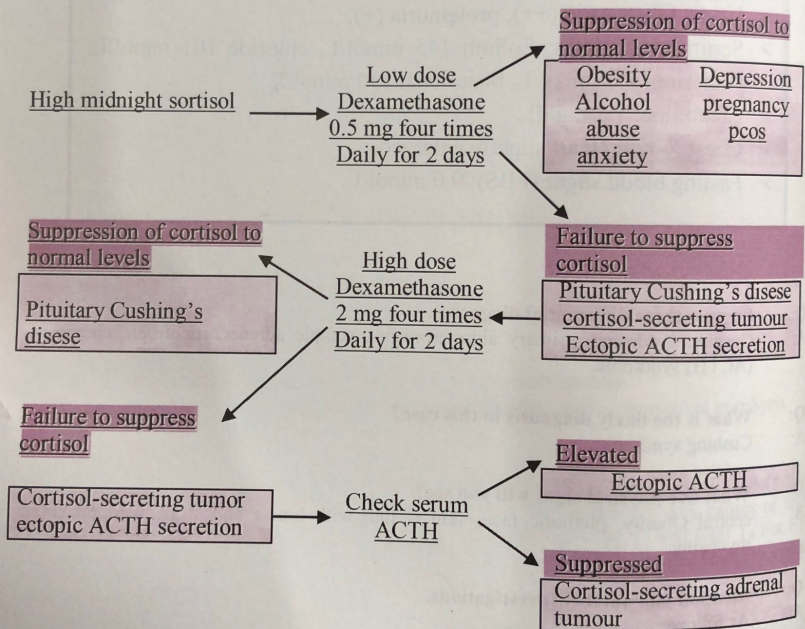
Q: Mention four further investigations.

A: As follows:

- Serum morning and midnight cortisol.
- Dexamethasone suppression test.
- Ultrasonography of abdomen (to see suprarenal gland)
- CT scan or MRI of suprarenal glands.

Key Points:-

Hypertension with hypokalemia and diabetes mellitus is highly suggestive of Cushing syndrome.



Case 36:-

A 25 year old female is complaining of excessive thirst, weakness, polyuria, nocturia and insomnia for 6 months. Her BP is 165/115 mmHg. Investigations reveals:

- Full blood count: Hb 12.7 g/dL, WBCs 6800/cmm, polymorphs 60%, lymphocytes 39%, monocytes 1%, ESR 20 mm in 1st hour.
- Urea: 38 mg/dL (normal 9-11 mg/dL).
- Creatinine: 1.2 mg/dL (normal 0.68-1.36 mg/dL).
- Serum electrolytes: Sodium 148 mmol/L, chloride 107 mmol/L, potassium 2.8 mmol/L, bicarbonate 32 mmol/L.
- RBS: 9.3 mmol/L. 16.7
- Chest X-ray: Cardiomegaly, left ventricular type.

Q: What is your diagnosis?

A: Primary hyperaldosteronism / conn's syndrome

Q: Suggest three investigations to confirm your diagnosis.

A: As follows:

- Serum aldosterone and rennin assay.
- 24 h urine potassium.
- CT scan or MRI or adrenal glands.

Key Points:-

Hypertension with hypokalemic alkalosis is highly suggestive of primary hyperaldosteronism; it is also called Conn's syndrome. It is and aldosterone secreting tumour responsible for <1% cause of hypertension. Excess aldosterone secretion leads to sodium retention, hypokalaemia and hypertension. It is due to adrenal adenoma in 60% (Conn's syndrome) and 30% bilateral adrenal hyperplasia. Serum renin is low in primary aldosteronism, whereas renin is high in secondary aldosteronism.

Treatment:- If adenoma, surgery. If hyperplasia, spironolactone 100-400 mg daily. For hypertension, amiloride and calcium channel blocker may be used.

Case 37:-

A 45 year old female presented with blood pressure of 170 / 100 mmHg. Investigation reveals:

- Na^+ : 156 mmol/L.
- K^+ : 2.5 mmol/L.
- Cl^- : 102 mmol/L.
- HCO_3^- : 30 mmol/L.

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Q: What are the abnormalities in the investigations?

A: As follows:

- Hypernatraemia.
- Hypokalaemia.
- Metabolic alkalosis.

Q: Name three possible causes of this investigation finding.

A: As follows:

- Cushing syndrome.
- Conn syndrome (primary hyperaldosteronism).
- Renal artery stenosis.

Q: Name four other investigation to confirm the diagnosis?

A: As follows:

- Plasma cortisol.
- Urinary free cortisol.
- Plasma rennin and aldosterone assay (to see the ration)
- USG of kidney and adrenal gland.
- CT or MRI of suprarenal gland.

Hypokalaemia with hypertension:-

- **Conn's syndrome** (primary hyperaldosteronism)
- **Cushing's syndrome**
- **Liddle's syndrome** (pseudo-hyperaldosteronism)
- **11-beta hydroxylase deficiency**
- **Carbenoxolone**, an anti-ulcer drug
- **Liquorice excess.**
- **Familial glucocorticoid remediable aldosteronism (GRA)**

Hypokalaemia without hypertension:-

- Diuretics
- GI loss (e.g. Diarrhoea, vomiting)
- RTA (type 1 and 2)
- Bartter's syndrome
- Gitelman syndrome

Case 38:-

A 22 year old man was admitted with the complaints of weakness and weight loss for several weeks. His blood pressure is 80/50 mmHg. Investigations reveals:

- Na^+ : 122mmol/L.
- K^+ : 6.3mmol/L.
- Cl_3^- : 20mmol/L.
- HCO_3^- : 30 mmol/L.

Q: What are the biochemical abnormalities?

A: As follows:

- Hyponatraemia.
- Hyperkalaemia.
- Metabolic Acidosis.

Q: What is the likely diagnosis?

A: Addison disease.

Q: Mention one important clinical sign.

A: Pigmentation of skin and mucous membrane.

Q: What are the common cause?

A: As follows:

- Autoimmune diseases .
- Tuberculosis.

Q: Name other four investigations to confirm the diagnosis.

A: As follows:

- Plasma cortisol and ACTH level.
- Synacthen test.
- USG of kidney and suprarenal gland.
- Plain X-ray abdomen (to see suprarenal calcification).

Key Points:-

In any patient with weakness, hypotension, pigmentation associated with low sodium and high potassium is highly suggestive of Addison disease. It is the primary adrenocortical insufficiency due to destruction of adrenal gland, resulting in deficiency in glucocorticoid and mineralocorticoid. Supine BP may be normal; but marked drop of systolic pressure on standing may occur. There will be low serum cortisol and high ACTH. Short synacthen test is helpful for diagnosis.

Treatment:- Hydrocortisone should be given, mineralocorticoid may be necessary. Steroid card should be maintained.

Case 39:-

A 19 year old woman was suffering from high grade fever, productive cough, shortness of breath, diffuse abdominal pain and vomiting .she is emaciated and dehydrated. Investigations reveals:

- Blood glucose: 28 mmol/L.
- Serum electrolytes: Na 118 mmol/L, Potassium 4.7 mmol/L, chloride 98 mmol/L and bicarbonate 8.1 mmol/L.
- Serum creatinine: 1.1 mg/dL.

Q: What is the likely diagnosis?

A: Diabetic ketoacidosis (DKA).

Q: What other investigations should be done?

A: Urine for ketone bodies.

Q: What are the complications?

A: As follows:

- Acute respiratory distress syndrome.
- Acute renal failure
- Thromboembolism due to dehydration .
- Cerebral oedema.
- DIC.

Key Points:-

Dry tongue, weight loss, high blood sugar with severe metabolic acidosis indicates DKA. It is common in type 1 diabetes mellitus. Three main problems in DKA are: (i) hyperglycaemia, (ii) hyperketonaemia and (iii) metabolic acidosis. Marked dehydration is common. Five percent cases develop coma in DKA. If bicarbonate is <12 mmol/L, it indicates severe acidosis.

Management of DKA :-**General management:-****(A). Therapeutic goals :-**

- 1) To restore the plasma volume and tissue perfusion .
- 2) To reduced blood glucose level and osmolarity towards normal
- 3) To correct acidosis
- 4) Replenish electrolyte losses.
- 5) Identify and treat the precipitating factors.

(B). Precipitating factors of DKA:-

- 1) Infections
- 2) CVA
- 3) MI
- 4) Pancreatitis
- 5) Alcohol abuse

General management:-

- 1) patient should kept N.P.O)
- 2) Gastric intubation in comatose patients to prevent vomiting and aspiration .
- 3) Indwelling catheter
- 4) Central venous pressure catheter in patients with pre-existing heart or kidney diseases to monitor subsequent fluid administration and evaluate the degree of hypovolemia .
- 5) Plasma glucose should be recorded hourly and electrolyte and PH atleast 2-3 hourly during the initial treatment period
- 6) patient should not receive sedative or opiod in order to masking signs and symptoms of impending cerebhal edema.
- 7) DVTporphylaxis inform of compression stockings.
- 8) Comprehensive flow chart that includes vital signs, serial laboratory data and therapeutic intervention

A. Fluid replacement C:- (total fluid deficit 4-5L)

- Initially 0.9% saline solution of choice to re-expand the contracted vascular volume as soon as possible.
- 1st liter → 1L/hour over first 1-2 hours
- After 2L of fluid have been given at the rate of 300-400 ml/hour .
- Use of 0.9% (normal) saline unless the serum sodium is > 150 mEq /L, then 0.45% (half) saline solution should be used.
- Give at least 3-4 L in 8-hours .
- Excessive fluid replacement > 5 L in 8 hours may contribute to acute respiratory distress syndrome or cerebral edema
- When blood glucose falls to approximately 250mg/dl , the fluid should be changed to 5% glucose-concentrating solution to maintain serum glucose in the range of 250-300 mg/dl .

B. Insulin replacement therapy :-

- First 0.1 unit/kg as a bolus to prime the tissue insulin receptors .
- Following the initial bolus , iv doses of insulin as low as 0.1unit/kg/hour continuously infusion or given hourly as an intramuscular injection.

Advantage of replacement of insulin deficiency :-

- Reduces the hyperosmolarity by reducing the hyperglycemia
- Decrease production of glucose by liver and increase utilization of glucose by peripheral tissue.
- Aim to reduce glucose concentration "50-70mg/dl/hours (target 250-300 mg per dl) if hyperglycemia not improve after initial first two doses of insulin , doubling the dose of insulin every 2-4 hours.

Overlap :-

- Once the DKA is controlled and patient is awake and able to eat S/C insulin therapy can be initiated .
- Bolus insulin approximately 0.6unit/kg or amount of insulin required in the previous 8-hours can also be helpful in estimating the initial insulin doses.
- low dose of i/v insulin will be needed for 30minutes to 1hours because of smooth transition from i/v insulin infusion to s/c bolus insulin

- Half of total daily dose can be given as long acting basal insulin and other half as a short acting premeal.)
- The increased insulin resistance is only present for few days and it is important to reduce both the basal and bolus insulin to avoid hyperglycemia
- A patient with new onset of type 1 DM usually still has significant beta cell function and may not need any basal insulin and only very low dose of rapid acting insulin before meal after recovery from the ketoacidosis .
- Patient with type 2 DM and DKA due to severe illness may initially required insulin therapy but can often transition back to oral agents during outpatient followup:

C. Potassium :-

- Total body potassium loss from polyuria and vomiting may be as high as 200 mEq.
- As acidosis is corrected , potassium flows back into the cells, and hypokalemia can develop if potassium replacement is not instituted.
- If patient is not uremic and has an adequate urinary out put, potassium chloride in dosage of 10-30 mEq/h should be infused during the second and third hour after beginning of therapy as soon as the acidosis start to resolve .
- ECG can be help in monitoring the patient's potassium status .
(high peaked T waves is sign of hyperkalemia)

D. Sodium bicarbonate :-

The use of sodium bicarbonate in management of DKA has been questioned because of following harmful consequences .

(i). Development of hypokalemia

(ii). Tissue anoxia

(iii). Cerebral acidosis

- It is recommended if arterial blood PH is 7.0 or less , one or two ampules of sodium bicarbonated (one ampule 44meq/50ml) should be added to IL of 0.45% saline with 20 mEq of kcl) .
- It can be repeated untill the arterial PH reaches 7.1 but should not be give if PH is 7.1 or greater because increase risk of rebound metabolic alklosis as ketones are metabolized .

Urea: BUN $\times 2.14$

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$2 \times \text{Na} + \text{serum glucose} + \text{urea}$
 $18 \quad 25$

BUN - Urea
 2.14

Case 40:-

A 70 year old lady was suffering from fever and frequency of micturition for 10 days. She became unconscious for the last 2 h. BP 90/60 mmHg, pulse 110/min, marked dehydration.

Investigation reveal:

- Full blood count: Hb 11.2 g/dL, WBCs 17,000/cmm, polymorphs 85%, lymphocytes 15%, platelets 2,10,000/cmm, ESR 12 min in 1st hour.
- Serum electrolytes: Sodium 162 mmol/L, chloride 110 mmol/L, potassium 4.6 mmol/L, bicarbonate 22 mmol/L.
- Serum creatinine: 1.4 mg/dL.
- Urine: Plenty of pus cells, albumin (++) , glucose (+++).
- Chest X-ray: Normal.
- CT scan of brain: Diffuse age-related cerebral atrophy.

Q: What is the likely diagnosis?

A: Hyperosmolar nonketotic diabetic coma with urinary tract infection (UTI).

Q: Suggest two investigations.

A: As follows:

- RBS.
- Urine for ketone bodies

Management of hyperglycemic hyperosmolar state :-

(hyperglycemia in the absence of significant ketosis)

Precipitating factors :-

- 1) Infections
- 2) MI
- 3) Stroke
- 4) Medications :- diuretics , corticosteroids , phenytoin

A. Fluid replacement:-

- Total fluid deficit (6-10L)

Choice of fluids:-

- If hypovolemia is present as evident by hypotension, oliguria, → fluid therapy should be initiated with 0.9% (normal) saline solution.
- In all other cases 0.45% (half) saline solution appears to be preferable as the initial replacement solution because the body fluids of these patients are markedly hyper osmolar 4-6L of fluid may be required in first 8-10 hours.
- Careful monitoring of patient sodium and water replacement.
- An important end Point of fluid therapy is to restore urinary output to 50 ml/hours.
- Once blood sugar level reaches 250mg/dl fluid replacement should be 5% dextrose in either water, 0.45% saline or 0.9% saline solution.
- Target glucose level 250-300 mg/dl to reduce the risk of cerebral edema.

B. Insulin :-

- Less insulin may be required to reduce the hyperglycemia in nonketotic patients as compared to those with diabetic ketoacidotic coma
- Infact fluid replacement alone can reduce hyperglycemia considerably by correcting the hypovolemia, which then increase both glomerular filtration and renal excretion of glucose
- Star insulin 0.05 units/kg/hour (bolus is not needed) and titrate to lower blood glucose level by 50-70 mg/dl/hour
- Once the patient has stabilized and the blood glucose falls to around to 250mg/dl, insulin can be given S/C

C. Potassium:-

- With absence of acidosis, there may be no initial hyperkalemia unless associated with (end stage CKD is present)
- There is less severe total potassium depletion than in DKA and less potassium replacement is therefore needed
- As results of insulin's effects on driving potassium intra-cellularly, it is recommended that potassium replacement be initiated earlier than in ketotic patients, assuming that now CKD or oligourea is present
- KCL (10 mEq per liter can be added to initial bottle of fluid administered if the patient's potassium is not elevated

Key Points :-

Hyperosmolar nonketotic diabetic coma (HNDC) may be the first presentation in diabetes mellitus. Common in elderly, Noninsulin-dependent diabetes mellitus (NIDDM), characterized by very high blood glucose (>50 mmol/L) and high plasma osmolality. No ketosis because insulin deficiency is partial and low insulin is present, which is sufficient to prevent ketone body formation, but insufficient to control hyperglycemia. Precipitating factors are large amount of sweet drinks, infection, steroid, thiazide, myocardial infarction, etc. plasma sodium is usually high. There may be high blood urea nitrogen (BUN); urea, creatinine and serum osmolality may be very high. Mortality rate may be up to 40%.

Case 41:-

A 52 year old factory worker was brought to the causality in a confused and drowsy state. He had been limping for 10 days. He had abdominal pain for the last 24 hours. He was afebrile, dehydrated and R/R of 18/min, B.P was 90/50. Abdomen was diffusely tender

- What three signs would you look for to arrive at a diagnosis?
- What four immediate investigations would be helpful in confirming the diagnosis.
- Give three important steps in management.

a). Following three signs should be sought:-

- Anemia, due to hemolysis and sideroblastic anaemia.
- Bluish line on gum margins due to deposition of lead supplied.
- Foot drop, wristdrop; motor neuropathy is characteristic of lead poisoning.

These features if present will strongly favors lead poisoning.

b). These are:-

- Haemoglobin with peripheral smear. This will show reduced haemoglobin with punctuate basophilia. (Basophilic stippling of RBC)
- Serum lead levels.
- RBC's porphobilinogen-deaminase and urinary porphyrinogen levels will be done. The former will be reduced, while latter will increase in case of acute intermittent porphyria.
- CT-brain to rule out concomitant organic lesion.

c)

- He will be admitted in hospital and will not be allowed to go back to the same workplace. In future occupational rehabilitation will be arranged.
- Intravenous sodium calcium ededate (EDTA) 75 mg/kg will be given for 5 days as a chelation therapy for lead poisoning.
- He will be hydrated. I.V line will be taken and normal saline 0.9% will be given with monitoring of intake and output.

Case 42:-

A 14 year old girl with type-I diabetes mellitus is brought with one day history of fever, altered consciousness and a single tonic clinic seizure. She is found to be confused and irritable. Temperature is 102°C , Pulse 96/min, B.P 110/75, no neck stiffness. No abnormality in the chest.

Investigations:- blood sugar 60mg/dl, urine sugar and ketone bodies are absent. CSF protein 70 mg/dl, sugar 45 mg/dl, cells 12/hpf mixture of polys and lymph's. Her condition failed to improve while her blood sugar rise to 125 mg/dl after I.V glucose.

- What three diagnoses would you consider?
- Suggest two investigations with their significance.
- Outline treatment of the most likely diagnosis

a). Would like to consider following three diagnoses:-

- Cerebral malaria** as she has 1 day history of fever, latered consciousness, no signs of meningeal irritation and hypoglycemia.
- Viral encephalitis**, suggested by fever, altered consciousness, fits, raised CSF proteins, elevated cell counts on CSF examination.
- Meningitis**, will be a suspected diagnosis due to fever, drowsiness, raised CSF proteins and cell count with mixture of polys and lymphocytes.

b). These are:-

- Giemsa stained thick and thin films will be made. This may show characteristic trophozoites of plasmodium falciparum, thus confirming cerebral malaria.
- MRI brain may show cerebral oedema especially enhanced signals in temporal lobes, characteristic of viral encephalitis.

c). Outline treatment of the most likely diagnosis:-

1. She will be admitted in high dependency unit.
2. I.V line will be taken.
3. Naso gastric tube as well as foley catheter will be passed.
4. Intravenous quinine dihydrochloride will be started. 20 mg/kg will be given over 4 hours, followed by 10 mg/kg, 8 hourly.
5. Monitoring of blood sugar will be done 2 hourly, as infection as quinine both can cause hypoglycemia.
6. Careful monitoring of fluid balance will be done.
7. Any evidence of superadded bacterial infection will be looked for, if needed, antibiotics will be given.
8. Severe anemia may need blood transfusion.
9. Monitoring of renal functions and hepatic functions will be done daily, as plasmodium falciparum can cause hepatic and renal failure.
10. ECG will be done daily as quinine causes prolonged QT interval.
11. She will be switched over to oral quinine as soon as she is able to take orally.

Case 43:-

A 64 years old man complained of lassitude and weakness of 6 months duration, together with occasional lumbar backache over the same time period. Three months prior to his admission he had been found to be anemic and had been treated as an outpatient with oral and parenteral iron. With symptomatic improvement during this time he has noticed nocturnal frequency. For 3 days prior to admission he had been oliguric. There was a past history of peptic ulceration and a long history of migraine headaches.

On examination he looked unwell and was pale. Blood pressure 180/120mmHg. R/R 28/min, a pericardial friction rub was present.

Investigations:-

Hb	8.6mg/dl
MCHC	33%
Chest	X-ray showed enlarged cardiac shadow and upper lobe venous distension.
Urine	Showed trace of albumin with 1-2 cells/hpf.

- Discuss five disorders in the differential diagnosis.
- Plan and justify your diagnosis
- Give steps in the management along with their justifications.

a). Five likely diagnoses can be:-

- Chronic renal failure due to diabetic nephropathy**, as he gives history of nocturnal with weakness and lassitude.
- Chronic renal failure due to obstructive uropathy** (stone / prostatic enlargement) this is suggested by occasional lumbar pain, followed by oliguria.
- Chronic renal failure due to multiple myeloma** as he is an elderly male, with generalized weakness, lassitude and anaemia. Nocturia and peptic ulceration can be due to hypercalcemia.
- Chronic renal failure due to analgesic nephropathy** as there is a long history of migraine and he might have taken various analgesics for a long period.
- Chronic renal failure due to chronic pyelonephritis** as he has history of lumbar pain.

b). Planning and justification of diagnosis:-

1. The most likely diagnosis is chronic renal failure due to NSAIDS induced tubulo interstitial nephritis. He has developed complications of uremia. That is uncontrolled hypertension, uraemic pericarditis, pulmonary edema and anaemia. There is past history of diabetes mellitus, fever, haematuria, pyuria and passing stones in the urine. But he does give history of migraine (taking NSAIDS) induced tubulo-interstitial nephritis is the third leading cause of intrinsic renal failure. Proteinuria (Traces) and presence of 1-2 white cells/HPF also supports the diagnosis. Other features may include white cells casts, red cell cast / HPF. Blood picture may show eosinophilia.

c). Management along with their justifications:-

1. He will be admitted in the ward.
2. I.V line will be taken.
3. Renal replacement therapy in the form of dialysis will be arranged. Regardless of the level of creatinine and serum potassium, he needs dialysis due to pericarditis and fluid overload.
4. There after intake output monitoring will be done.
5. Echocardiogram will be done to check for cardiac tamponade or pericardial effusion. Pericardiocentesis will be planned in case of clinical deterioration.
6. Control of blood pressure is important in addition to dialysis he will be given antihypertensive like calcium channel blockers and alpha blockers along with diuretics.
7. He is anaemic Improvement in hemoglobin levels will help to improve his weakness. Moreover it will reduce the fluid overload. He will be started on s/c erythropoietin once weekly basis. Intravenous iron will be given if required.
8. Protein-restricted diet will be given. Phosphate binders (calcium carbonate or acetate) will be given in case of hyperphosphatemia.

Indication for dialysis in renal failure :-

- Fluid overload not responding to diuretic therapy
- Uremia
- Metabolic acidosis
- Uremic pericarditis
- Uremic gastritis

A B C D E

Causes of anemia in CKD :-

- Deficiency of erythropoietin
- Toxic effects of uremia on BM
- Reduced survival of RBC's
- Blood loss due to capillary and poor platelets function
- Reduced intake, absorption and utilization of dietary iron.

Case No. 44:-

A previously healthy 30 year old young gentleman was found to have raised ALT (twice normal) during routine investigations. Repeated LFT's show persistent raised ALT (around twice normal)

- What are the five possible causes for his raised ALT?
- Name five investigations that you would advise.
- What precaution would you tell him before a final diagnosis is reached.

a. The five possible causes of his raised ALT are:-

- Chronic viral hepatitis.
- Wilson's disease.
- Alcoholic liver disease.
- Non-alcoholic steato hepatitis (NASH)

a1: differential diagnosis of ALT > 1000 :-

- Acute viral hepatitis
- Ischemic hepatitis
- Drug induced hepatitis

b. These investigations include:-

- Viral serology; anti HCV antibodies and HBsAg along with Anti HBcIgM and IgG antibodies, 24-h urinary cu excretion
- Repeat LFT's will be done to look for AST, alkaline Phosphatase and Reverse ALT: AST ratio > 2 (alcoholic liver disease), which is typical of alcohol liver disease along with raised gamma GT.
- ANA and Anti LKM antibodies for autoimmune hepatitis.
- Ultrasound abdomen to look at the hepatic echotexture, size and width of biliary channels, portal vein or any evidence of splenomegaly or ascites.
- Liver biopsy, to look at the degree of inflammation, fibrosis and characteristic histopathological changes of the underlying disorder.

c. The patient will be advised to discontinue alcohol if he is taking it:-

- If he is obese then he will be advised to reduce weight.
- He will be asked to avoid hepatotoxic drugs.
- Use of disposable sterilized syringes and screening of blood before transfusion will be advised for the future.
- Screening of the other family members will be done.

Case No. 45:-

A thirty six year old man presents with a long history of low grade fever, sinus discharge on many occasions hemoptysis and difficulty in breathing since about 5 years. He had received broad spectrum antibiotics and a full course of ATT, without benefit. On examination, temperature 99.6°F, resp rate 24 per minute, B.P 110/70 mmHg and pulse 100/min. nasal septal perforation. Coarse crepitation's and bronchial breathing over mid thorax posteriorly. On presentation, patient had following investigations. Urine D/R albumin ++, RBC-plenty, pus cells - nil: Chest x-ray revealed two big thick walled cavities in each lung in mid radiological zones.

a. Name the 4 lab investigations and justify how these will help you to reach an appropriate diagnosis and subsequent management.

b. what is management ?

ESR, CRP, complement level

a. Following 4 lab investigations are required to reach a diagnosis:-

- 1 **P-ANCA and C-ANCA:** these anti-neutrophil cytoplasmic antibodies help in the diagnosis of small vessel vasculitis including churg-strauss syndrome, microscopic polyangitis and Wegener's granulomatosis. C-ANCA or anti-proteinase 3 antibodies has a high specificity for Wegener's granulomatosis (90%)
- 2 **Transthoracic lung biopsy** has the maximum yield. It will show characteristic histologic features of Wegener's granulomatosis. These are vasculitis, granulomatous inflammation, geographic necrosis and acute/chronic inflammation.
- 3 **Renal biopsy** will demonstrate segmental necrotizing glomerulonephritis.
- 4 **S/creatinine and blood urea** to demonstrate renal insufficiency. Once proteinuria and haematuria develops, further renal deterioration is quite rapid.

b. Management:-

- He will be admitted in the ward.
- He will be started oral steroids, prednisolone, 1mg/kg along with cyclophosphamide. Cyclophosphamide will be continued for 3 months and then it will be substituted by methotrexate or azathioprine, for at least 12-15 months.
- renal function will be continuously monitored and acute renal failure will be treated with dialysis.

Case No. 46:-

A 40 year old lady presented in OPD with history of generalized weakness low grade fever and easy fatigability of 4 years. She also reports of decrease in appetite, significant loss of weight with constant nausea over the past 16 months. She also insists that her skin colour was fair which have recently changed to black over the body sparing hands and feet. Her past family history is insignificant.

Vital signs temperature 99°F, pulse 88/min. BP 85/50 mmHg, respiratory rate 22/min. Dark brown pigmentation is present all over the body, especially over the elbows and skin creases. Rest of the clinical examination is normal.

- a. What is diagnosis ?
- b. what is important investigations ?
- c. what is treatment ?

a. She is most likely suffering from adrenal insufficiency

b. The investigate her disease following lab tests are required:-

- 1 **Complete blood count:** will show anaemia of chronic disease, neutropenia, lymphocytosis and moderate eosinophilia.
- 2 **Serum electrolytes:** hyponatremia (90%) , hypoglycemia and hyperkalemia (65%) is characteristic of Addison's disease.
- 3 **Short synacthen test:** 0.25 mg of synthetic ACTH will be given parentally. Then estimation of serum cortisol is done between 30-60 minutes. Serum cortisol less than 20 ug/dl indicates Addison's disease.
- 4 **Plasma ACTH levels** will be elevated due to loss of feedback inhibition.
- 5 **Anti-adrenal antibodies** are elevated in 50% of cases of autoimmune adrenal insufficiency. This will help to ascertain the likely cause of adrenal insufficiency.
- 6 **CT abdomen** may show small non-calcified adrenal in autoimmune Addison's disease, while adrenals are enlarged due to granulomatous or metastatic disease. Intra-adrenal calcification is seen in case of tuberculosis

C. MANAGEMENT:-

- 1 ➤ Treat the infections
- 2 ➤ Correct the Dehydration
- 3 ➤ She will be started glucocorticoids either hydrocortisone (15-25 mg) or prednisolone (2-3 mg) daily. Two third will be given in the morning and one third in the evening. Further adjustments in dose will be done after the clinical response.
- 4 ➤ Fludrocortisone acetate is given as a part of mineralocorticoid replacement.
- 5 ➤ She will be advised to wear a medical alert bracelet, indicating that she is taking glucocorticoids for adrenal insufficiency.
- 6 ➤ Treatment of underlying cause .

Case No. 47:-

A 30 year old man was brought to outpatient by his friend 10 days prior admission. He developed high grade fever with chills and evening rise. He had cough with scanty sputum in last 2 months. Fever did not settle despite various antibiotics and antimalarial therapy. He had to miss his annual business trip to South Africa due to his illness. He has never smoked. On examination he was pale, ill looking with temperature 39.2°C. Pulse was 120/min, regular, B.P was 120/80 mmHg. He had white coated tongue and cervical adenopathy. Chest examination was normal. Cardiac auscultation revealed pericardial rub. Other systems were normal.

Investigations:-

Hb	108g/dl
ESR	60 mm in first hour.
TLC	5.2 x 10 ⁹ /L
Neutrophils	88%
Lymphocytes	10%
Eosinophil's	2%

CXR showed enlarged cardiac shadow with b/l hilar adenopathy

- List your differential diagnosis in order of priority.
- How will you investigate this patient?

a. D/D of fever + cervical lymphadenopathy :-

1. Lymphoma
2. Tuberculosis
3. Infectious mononucleosis
4. HIV seroconversion
5. Toxoplasmosis
6. Syphilis
7. Sarcoidosis
8. Brucellosis

b. Following investigations will be sent :-

- 1 ➤ Sputum for AFB, to look for mycobacteria tuberculosis.
- 2 ➤ Bronchoscopy followed by bronchial washings these washings will be subjected for ZN staining, C/S and malignant cells.
- 3 ➤ In sarcoidosis, there is reversal of CD_4 to CD_8 ratio.
- 4 ➤ Excision biopsy of cervical lymph nodes. It will show the characteristic histological changes of lymphoma, tuberculosis, sarcoidosis.
- 5 ➤ CT scan chest followed by CT guided fine-needle aspiration of hilar lymph nodes. This will not only help us to determine the stage of lymphoma but will also enable us to rule out other causes.
- 6 ➤ Ultrasound abdomen to look for hepatosplenomegaly or intra-abdominal lymph nodes.
- 7 ➤ Peripheral smear examination to look for atypical lymphocytes seen in infectious mononucleosis, HIV and toxoplasma.
- 8 ➤ VDRL will be done to rule out syphilis.
- 9 ➤ HIV enzyme linked immunosorbent assay.

Case No. 48:-

A 65 year old male, smoker for 10 years presents in casualty department with severe shortness of breath and unconsciousness for 12 hours. On enquiry, his son informs that his father had persistent cough and progressively increasing shortness of breath for the last 4 years.

On examination, pulse is 90/min, B.P is 130/75 mmHg. He has moderate clubbing and cyanosis. Respiratory rate 30/min, chest expansion less than 1cm and bilateral basal crepitations.

X-ray chest shows loss of volume and honeycombing appearance.

- Give the most likely diagnosis and justify it.
- Discuss 4 important investigations useful in diagnosis and management this case.
- Discuss 4 important steps in the short and long term management.

a. The most likely diagnosis is cryptogenic fibrosing alveolitis :-

Essential of diagnosis :-

- The most common of the diffuse interstitial pneumonia is pulmonary fibrosis associated with histopathological pattern of UIP (usual interstitial pneumonia), when no associated cause is evident, called IPF

Diagnosis of IPF :-

- 1 ➤ Age > 65 years
- 2 ➤ Idiopathic disease on history and B/L end-inspiratory crackles un-altered with coughing.
- 3 ➤ Restrictive physiology on lung function tests
- 4 ➤ Diffuse, patchy fibrosis with pleural based honeycombing

Clinical features :-

- 1 ➤ Progressive shortness of breath.
- 2 ➤ Persistent dry cough.
- 3 ➤ Clubbing and cyanosis.
- 4 ➤ Reduced chest expansion.
- 5 ➤ B/L basal crepitations unaltered with coughing

b. investigations:-

- 1 Arterial blood gases will show arterial hypoxemia with normal or low PaCo₂. This is due to alveolar capillary block and ventilation-perfusion mismatch.
- 2 High resolution CT chest will show characteristic reticular shadowing and ground glass opacification.
- 3 Bronchoalveolar lavage may show increased number of macrophages and neutrophils.
- 4 Transbronchial lung biopsy, to rule secondary cause of pulmonary fibrosis and determine the type of cryptogenic pulmonary fibrosis.

c. Management:-

General management :-

- 1 ➤ Smoking cessation
- 2 ➤ Oxygen therapy by face mask
- 3 ➤ Ventilatory support
- 4 ➤ Counselling regarding domiciliary oxygen therapy
- 6 ➤ Cortisteroids / immunomodulator / immunosuppressant are not recommended

Specific management :-

- Perfinidone and nintedinib

Case No. 49:-

A young lady with postpartum fever for one week presented in emergency department with H/O bleeding from various sites and oliguria. On O/E, she is anaemic with cutaneous bleeding and mildly jaundiced. There is no hepatosplenomegaly. Investigation revealed Hb: 8.0 gm/dl, TLC 6000/mm³, T/bilirubin 3mg/dl with direct bilirubin 1 mg%, ALT 100 IU, LDH 1230 IU, serological test for B,C and E are negative. Her blood urea is 200mg%.

- a. What is the most likely diagnosis?
- b. List the lab abnormalities in this condition.
- c. What are treatment options?

a. The most likely diagnosis is haemolytic uremic syndrome. This is indicated by postpartum fever, oliguria, bleeding from various sites, raised indirect bilirubin due to haemolysis, raised blood urea and LDH levels.

It causes microangiopathic hemolytic anaemia, thrombocytopenia and uremia.

b. investigations :-

- 1> Platelet count; will be low.
- 2> Fragmented RBC's will be seen on peripheral blood smear.
- 3> Low haemoglobin.
- 4> Raised blood urea and creatinine.
- 5> Raised LDH.
- 6> Indirect hyperbilirubinaemia.
- 7> Raised Urinary urobilinogen in the absence of bilirubinauria.

c. Plasmaphoresis, 60-80 ml/kg will be done.

Case No. 50:-

A 17 years old girl is admitted with increasing fatigue and pallor for 3 months. There is no history of chronic blood loss. She has not taken any medicines. On examination, she is markedly pale, with hepatosplenomegaly, her investigations show; Hb 8.0 gm/dl, normal MCV and normal TLC, DLC and platelet count.

- a. what is differential diagnosis ?
- b. what is investigations?

a. D/D of normocytic normochromic anemia with hepatosplenomegaly:-

- 1 Lymphoreticular disorder e.g lymphoma.
- 2 Aleukemic leukemia.
- 3 Haemolytic anaemia, which could be either due to intrinsic defect (hereditary spherocytosis) or extrinsic defect (autoimmune haemolytic anaemia).
- 4 Chronic liver disease.
- 5 Disseminated tuberculosis.

Investigations:-

- 1 **Peripheral blood** smear, may show blast cells (Leukemia), leucoerythroblastic picture (bone marrow infiltration due to lymphoma, leukemia), spherocytosis, elliptocytes, fragmented RBC's.
- 2 **Chest x-ray** Chest x-ray, may show hilar / mediastinal adenopathy due to lymphoma or tuberculosis. If these are seen, then trans bronchial or transthoracic lymph node biopsy will be done to confirm diagnosis. Chest x-ray may also show upper lobe consolidation, capitations or fibrosis due to tuberculosis.
- 3 **Bone marrow aspiration and biopsy** will be done to confirm the causes of anaemia. It will show the exact cause of her pallor.
- 4 **Reticulocyte count** will be elevated in case of hemolytic anaemia, while conditions causing bone marrow suppression leads to low reticulocyte count.
- 5 **Ultrasound abdomen** to look at the texture, size of liver, spleen and portal vein. It may show intra-abdominal lymph nodes, ileocaecal mass due to tuberculosis or ascites, which may be due to tuberculosis, liver disease or lymphoma.

Case No. 51:-

A 20 year old labourer was brought in examination department with few hours history of nausea, vomiting, and abdominal pain and decreased vision after consumption of a drink. On examination he was drowsy and breathing heavily. Pupils were dilated and sluggishly reacting to light. Fundoscopy revealed hyperaemia of optic discs and evidence of retinal edema. Rest of the examination was normal. Subsequently his conscious level deteriorated and he developed generalized tonic, clonic fits. Labs ABG's, $\text{pH}=7.29$, $\text{P}_a\text{O}_2=89\text{mmHg}$, $\text{P}_a\text{CO}_2=30\text{mmHg}$, $\text{HCO}_3^- 8\text{meq/L}$

- a. What is your diagnosis?
 - b. How would you manage this case?
- a. The patient most likely has methanol poisoning. History of drink, followed by drowsiness, decreased vision, poorly reactive dilated pupils, metabolic acidosis with normal sugar and urea suggests the diagnosis.
 - b. Methanol is metabolized to formaldehyde, which cause accumulation of hydrogen ions leading to toxicity. So inhibition of methanol metabolism is important and is carried out by the administration of ethanol or fomepizole. These inhibit alcohol dehydrogenase. Ethanol is given as I.V. infusion until no methanol is detected in the blood.
 - To remove methanol and formaldehyde, hemodialysis will be carried out.
 - To prevent ocular toxicity, 30 mg of folic acid will be given. This will accelerate formaldehyde metabolism.
 - Supportive measure to combat metabolic acidosis, fits, hypocalcaemia and shock will be carried out.

spec
 → Ethanol
 → fomepizole
 → Dialysis
 → folic acid

→ Bicarb
 → Hypocalcaemia correct
 → Control seizures
 → Electrolyte imbalance correct
 → shock.

Case No. 52:-

A 40 year old male presented with dyspnea, cough and hemoptysis of 10 days duration. He has never smoked. On examination, temperature 37°C, pulse 110/min, B.P. 170/100 mmHg, pitting ankle adema is +ve, JVP is not raised. On auscultation of chest, there are B/L crepitations, CVS examination is unremarkable.

Lab investigations: Hb 10.0g/dl WCC $16 \times 10^9/L$

ESR 85 mm/1st hour, microcytic hypochromic picture, urea=230mg/dl, creatinine 8.5mg/dl, urine R/E is albumin ++, numerous RBC, granular cast. 3-4 X-ray chest: wide spread interstitial shadowing of both lung fields. ABG: P_aO_2 58 mmHg, P_aCO_2 20mmHg.

- a. What is your differential diagnosis?
- b. How will you further investigate this case?
- c. How would you manage this patient?

Ans

a. **D/D diagnosis of reno-pulmonary syndrome :-**

- 1 Good Pasture syndrome.
- 2 Wegener's granulomatosis.
- 3 Microscopic polyangitis.
- 4 Atypical pneumonia due to Mycoplasma or legionella, causing renal involvement too, in the form of acute nephritic syndrome.

b. **Investigations:-**

- 1 Anti-GBM antibodies will be present in Good Pasture syndrome.
- 2 ANCA antibodies will be done p-ANCA is present in case of microscopic polyangitis while c-ANCA is raised in Wegener's granulomatosis.
- 3 Sputum C/E.
- 4 Serological tests for Mycoplasma IgM and Legionella antibodies.
- 5 Urinary Legionella antigen assays will be done.
- 6 Renal biopsy may demonstrate glomerular sclerosis with or without crescent formation and liner deposition of IgG and C3 along the basement membrane in case of Good-Pasture syndrome. Wegener's and microscopic polyangitis are small vessel vasculitis, with granuloma formation in Wegener's granulomatosis.

c. Management:-

- 1 He will be managed in high dependency unit.
- 2 O2 inhalation (4-5 L/min) will be started.
- 3 Plasma exchange will be carried out as Good-Pasture / anti-GBM glomerulonephritis is the most likely cause. This modality of treatment has better outcome in ANCA positive vasculitis as well.
- 4 Steroids will be given to suppress inflammation.
- 5 Immunosuppressant e.g. cyclophosphamide will be started to inhibit further antibody synthesis.
- 6 As he already has advanced acute renal failure, so he will be started on haemodialysis as well to remove uraemic toxins. This can be scheduled according to serial estimation of blood urea of creatinine.
- 7 Monitoring of ABG's, serum potassium, urea, creatinine and blood pressure will be carried out.

Case No. 53:-

A 62 year old man has presented with progressive deterioration of vision in the left eye for the past 3 months. On fundoscopic examination, left disc revealed pallor and reduced vascularity but right fundus showed papilledema and dilated veins.

- a. What is the differential diagnosis?
- b. What three relevant investigation you will carry out?

a. differential diagnosis:-

1. Optic atrophy may follow long standing papilledema which may be due to
 - a. Raised intracranial pressure.
 - b. Raised blood pressure.
 - c. Central retinal vein thrombosis.
2. Demyelination : Multiple sclerosis causes optic atrophy.
3. Metabolic cause e.g. B12 deficiency.
4. Large vessel vasculitis (giant cell arteritis)

b. Investigation:-

1. CBC (high HB , RBC counts high (polycythemia) , HB dec , MCV inc (vitamin B 12 deficiency , inc ESR (Gaint cell arteritis)
2. CT/MRI brain (features of SOL, periventricular demyelinating plaque (MS) .

→ IC space occupying lesion
→ optic neuritis
→ Glaucoma
Optic nerve compression

Case No. 54:-

A 35 year old farmer is brought to you in emergency department with history of snake bite 3 hours ago. On physical examination pulse rate 80/min, temperature 99°F, B.P. 100/70 mmHg with grossly swollen left legs. Systemic examination is normal.

- a. Discuss steps of management with their justification.
- b. What investigation will you like to do in emergency and why?

a.

- Patient and the bitten part will be immobilized.
- Minimal handling of the bitten area will be done.
- Ice and tourniquet application will not be done.
- 46 vials of antivenom will be given in 250-500 ml of saline, slowly intravenously.
- Tetanus immunoglobulin with tetanus toxoid will be given.
- As he has fever and his leg is grossly swollen so antibiotics with gram positive cover will be given.
- Repeated doses of 2 vials every 6 hours for up to 18 hours will be given.
- I/V line will be maintained and adequate hydration will be ensured to maintain good urinary output.
- Diazepam for anxiety.

b.

- 1 CBC: (TLC increases in cellulitis, thrombocytopenia in DIC)
- 2 P-film: fragmented RBC's
- 3 Coagulation profile: PT, APTT, INR and FDP ins in DIC
- 4 Blood grouping and cross-matching
- 5 Serum urea and creatinine, to look for determining renal function due to acute tubular necrosis secondary to snake venom.
- 6 Urine C/E to look for haematuria.

Case No. 55:-

A bus driver of 30 years of age consults the doctor with history intermittent fever, body aches, and feeling unwell for the last 3 weeks. On examination temperature 38°C, several slightly tender enlarged cervical lymph nodes are palpable. In the left groin some enlarged lymph nodes are noted. The rest of the examination was normal. Investigation showed Hb 12.5 gm%, ESR 40/1st hour, WCC 6×10^9 /L, peripheral film shows abnormal mature monocytes serum bilirubin 1mg%, ALT 80 I.U. serum alkaline Phosphatase 280 I.U. X-ray chest is normal.

A. Give five differential diagnosis.

B. What are the five most useful investigations?

a. Differential diagnosis :-

- Infections (infectious mononucleosis, CMV, HIV seroconversion)
- Malignancy (lymphoreticular malignancy)

b. investigations:-

- 1 Heterophil antibody test; usually positive within 4 weeks of onset of illness (Infectious mononucleosis)
- 2 HIV antibody by ELSIA.
- 3 PCR for EBV / CMV on blood.
- 4 Lymph node biopsy; so as to rule out lymphoproliferative disorder.
- 5 Bone marrow examination to rule out infiltration with lymphoma cells.

Case No. 56:-

A 19 year old student is seen in examination department with 3 days history of weakness of both legs. About 2 weeks ago, he had influenza like illness. For the last 2 days, he had developed dropping of both eyelids. He also feels tingling in fingers. Physical examination reveals left sided ptosis, slight b/l facial weakness. Rest of the cranial nerves are normal. In the limbs, there is moderate weakness, more pronounced distally. Sensations are normal. Left biceps reflex is present. Rest of the muscle stretch reflexes are not elicitable.

- What is the most likely diagnosis?
- Write two relevant investigations.
- Write three treatment modalities.
- What is the prognosis?

a)

It is Guillain-Barre syndrome, an acute demyelinating polyradiculoneuropathy. The preceding history of influenza like illness followed by progressive ascending pattern of weakness of the body, also involving the face and characterized by areflexia suggests the diagnosis.

b) Investigations :-

- CSF examination, will reveal high protein content with normal cell count (albuminocytological dissociation)
- Nerve conduction studies; will reveal demyelinating polyneuropathy.

c) Management :-**1. General management:-**

- Admitted the patient in ICU
- Continuously monitoring pulse, oxygen saturation, postural blood pressure, FVC and chest expansion
- Chest physiotherapy
- Care of skin, bowel, bladder and paralysed limb
- DVT prophylaxis in form of compression stocking and low dose heparin

2. Specific management :-

- IVIG 40 mg/kg for five days
- Plasmapheresis starts within 7 days of onset of symptoms in ambulatory patients and within 30 days of onset of symptoms in non-ambulatory patients

d)

Most patients make good recovery, but this may take many months and 10-20% of patient are left with persisting disability.

Case No. 57:-

A 55 years old male patient is admitted with history of generalized tonic clonic seizures. He is dysphasic but his son gave a history that he has not been well for past 4 days with high temperature and had been confused since one day before admission. Appendectomy was done 5 years ago, he was treated for mild hypertension for 5 years. On examination he was febrile (temp. 102.0°F), restless and disoriented. He had fluent dysphasia, possibly right homonymous hemianopia. Investigation showed Hb 13.5 gm%, WBC 10.5x10⁹/L, and ESR 50 mm in 1st hour.

- Give 2 possible diagnosis?
- What 4 investigations are required to reach the diagnosis?
- What treatment is available for this condition?

a)

- Viral encephalitis; preceding history of fever, confusion followed generalized tonic-clinic seizures suggest the diagnosis.
- Brain abscess; history of fever, fits with focal neurological deficit raises the doubt of space occupying lesion, most likely brain abscess.

b)

- CT and MRI brain:** may show diffuse areas of oedema, often in temporal lobe in case of encephalitis. Ring enhancing hypodense lesion will be seen on CT contrast in case of brain abscess.
- EEG:** shows characteristics slow wave changes in encephalitis.
- CSF examination:** will reveal raised protein, normal glucose and lymphocytosis in encephalitis.
- Specific viral blood and CSF serology** (for herpes simplex virus) will help confirm the diagnosis of viral encephalitis as well.

c) management:-**1. general management :-**

- Pass NG tube
- Pass foley's catheter
- Caloric fluid-electrolyte balance
- Care of skin, bowel and bladder

2. Specific management :-

- He will be treated immediately with intravenous acyclovir (10mg/kg 8 hourly). This will be given for 10 days.
- Seizures will be treated with anticonvulsants.

Case No. 58:-

A 50 years old man who is diabetic and hypertensive for last 10 years complain of drooping of eyelids and diplopia for last six months. He is on propranolol, hydrochlorothiazide and glibenclamide. His condition worsened during a recent bout of chest infection. On examination he had normal pupil with normal tendon reflexes and showed no other focal neurological deficit.

- What is the likely diagnosis?
- What further clinical observations could substantiate the diagnosis?
- How will you confirm the diagnosis?
- Give an outline of management.

a)

- The patient is most likely suffering from myasthenia gravis. The features suggesting it:-
- b/l ptosis and binocular diplopia in the presence of normal pupil.
- Normal tendon reflexes.
- No neurological deficit.
- Worsening of symptoms during bout of chest infection.

b)

1. fatigability tests
2. extra-ocular movements (binocular diplopia)
3. proximal muscle weakness
4. normal deep tendon reflexes

c)

- the diagnosis is confirmed by edrophonium test,
- single nerve fiber Electromyography shows decremental response and jitters and block
- antibodies (acetylcholine receptor antibodies elevated in 90% of cases and anti Musks antibodies)
- CT chest (To exclude co-existing thymoma)

d) management :-

- Long acting anticholinesterase pyridostigmin (amygra) 60mg x 4 times daily.
- Immunosuppression with azathioprine 2-3 mg/kg/day.
- Thymectomy should be considered in all patients younger than 65 years of age unless weakness restricted to extra-ocular muscles.
- **Indication for corticosteroids (1mg/kg/day)**
- Poor respond to anticholinesterase medications
- Exacerbation of weakness
- Respiratory failure

❖ **IVIG / plasma pheresis**

- 1) Myasthenia crisis
- 2) Stabilizing the patients before thymectomy

❖ **Invasive / non-invasive Ventilation**

- 1) Myasthenia crisis

❖ **Difference between myasthenia crisis and cholinergic crisis**

- 1) Myasthenia crisis due to insufficient medications or drug resistance leading to worsening of myasthenic weakness
- 2) Cholinergic crisis due to excess of cholinergic medications leading to salivation, lacrimation , miosis .

❖ **Factors responsible for myasthenia crisis**

- 1) Noncompliance with medications
- 2) Infections
- 3) Psychological stressor

❖ **Important associations**

- 1) Thymic hyperplasia
- 2) Thymoma
- 3) Other autoimmune diseases (SLE, RA and thyroid disorders)

❖ **Drugs my exacerbate myasthenia**

- 1) Antibiotics (macrolides , quinolones , aminoglycosides , tetracycline and chloroquine)
- 2) Antidysarrhythmic drugs (beta blocker , clacium ,chammel blocker , procainamide and quinidine)
- 3) Antipsychotic (phenothiazine)

❖ **Dafferential diagnosis of ptosis with meiosis**

- 1) Horner syndrome

❖ **Dafferential diagnosis of ptosis with dilated pupil**

3rd nerve palsy

Case No. 59:-

A 25 year old female presented with history of palpitation, excessive sweating, weakness with frequent bowel movements. She gave history of weight loss despite of good appetites. On examination, skin is warm and moist, pulse 90/min, BP 150/60 mmHg. On auscultation systolic murmur is audible. In laboratory investigations Hb is 11.5 gm%, ESR 65mm.1st hour. Urine examination is positive for pregnancy test.

- a. What is the diagnosis?
- b. What investigation will you carry out?
- c. What is the treatment?

a)

She is suffering from hyperthyroidism as she has palpitations, sweating, diarrhea, and weight loss with normal appetite. On examination she is tachycardia, has warm and moist skin. All these features suggest hyperthyroidism as the most likely diagnosis.

b)

- 1 Free T4 and T3 will be elevated.
- 2 Supersensitive TSH will be decreased as a part of negative feedback effect.
- 3 TSH-R Antibodies, will be elevated in case of graves' disease.
- 4 Anti-thyroglobulin or thyroidperoxidase antibodies, will also be elevated in case of graves' disease.
- 5 Serum β HCG levels.

c)

- 1st trimester (PTU 200-300mg)
- 2nd trimester (carbimazol 20-30mg)
- Breast feeding (PTU 200-300mg)
- Propranol 60mg twice or thrice daily
- Thyroid surgery (2nd trimester)
- Block and replace therapy is not recommended
- Radio-iodine is not recommended

Case No. 60:-

A 60 years old smoker having frequent bouts of cough reported to emergency department with severe dyspnea after an episode of cough and chest pain. He is cyanosed, has pulse of 110/min, resonant barrel shaped chest with wheezing and diminished breath sounds on the left.

- What is most likely diagnosis?
- What are the three most important investigations?
- How are you going to manage this patient?

a)

As the patient is a smoker and he has developed acute shortness of breath after an episode of cough and chest pain. He also has diminished breath sounds on the left side. Considering all this, the most likely diagnosis is left sided pneumothorax with underlying chronic obstructive pulmonary disease (COPD)

b) Investigation:-

- **Chest x-ray PA view :** will demonstrate hyper inflated right lung, with collapsed left lung and associated pneumothorax.
- **Arterial blood gases:-** may reveal hypoxemia, acute respiratory alkalosis.
- **Echocardiography:-** shows EF and pulmonary artery pressure
- **ECG:-** shows tachycardia , Rt-axis deviation , diminution of QRS complex voltage and T-wave inversion .

c. Treatment of COPD:-

- General measures
- Elimination of long term exposure of tobacco smoke , combustion of biomass fuel and inhaled toxins
- Annual vaccination of H, influenza and pneumococcal

Indication for treatment:-

- $FEV_1 < 50\%$ of predicted (Gold class III / IV)
- More then two exacerbation in previous one year
- One or more hospitalization stay in previous years

Oxygen therapy :-

- If resting hypoxemia $\text{PaO}_2 < 56$ mm of Hg
- Continuous oxygen supplement 24 hour per day

Inhaled bronchodilators :-

- SABA (albuterol, salabutamol) + SAMA (ipratropium bromide)
IPRATEC-S
- LABA (formetrol) + LAMA (tiotropium) TIOVAIR-F
- LABA (formetrol) + ICS (budesoid)..... COMBIVAIR

Corticosteroids:-

- Oral corticosteroids are not recommended in COPD
- Inhaled Corticosteroids always used in combination with LABA

Theophylline:-

- Is indicated if patient not responding to LABA+ICS

Antibiotics :-

- Indicated in (I) acute exacerbation (II) acute bronchitis (III) prevention of acute exacerbation of chronic bronchitis
- Antibiotic used in patients of COPD (I) macrolides (II) quinolones (III) amoxicilline + clavulanate

Phosphodiesterase inhibitors :-

Roflumilast

Case No. 61:-

A 60 years old woman presented with a 6 months history of general ill health, constipation, and backache, difficulty in walking, dizzy spells and sore eyes. The patient gave past history of falling in the bathroom after a dizzy spell one year back and of recurrent attacks of pain in the flanks for the past 18 months. Washing hands with cold water makes her very uncomfortable. Examination reveals a pleasant educated lady who looks slightly pale. She has a temperature of 99°F, her eyes are red looking, B.P is 160/90mmHg. Her pulse is 80/min and a soft systolic murmur is audible at the left sternal edge. She has brown looking painless skin lesions on the tip of her big toes bilaterally. Wasting of the thigh but the ankle and knee jerks are brisk and her planters seem to be upgoing. She can walk, through with some difficulty. A few exudates are visible in both discs. A soft spleen is 2cm palpable. She has hemoglobin of 10mg, TLC of 3500 with a normal differential count and an ESR of 80 mm in the first hour, urine shows ++ proteins.

Vertebral column seems to be osteoporosis, and T8 vertebra is partially collapsed.

Handwritten notes:
 CRAB — Renal
 Calcinosi Bone lesion
 AL amyloidosis

- What is the most likely diagnosis?
- Give three differential diagnosis and rationale for each.
- What investigations would you do to confirm the diagnosis? Give the finding that you will expect in these investigations?

a)

- The most likely diagnosis is multiple myeloma. The reasons for making diagnosis include:-

Essential of diagnosis :-

- Dizzy spells due to hyper viscosity, anaemia.
- Pain in flanks due to renal stones.
- Brown, painless skin lesions, tophi, due to hyperuricaemia.
- UMN type of paraplegia due to vertebral column involved, confirmed on x-rays.
- Spine, which show osteoporotic spine with partially collapsed T8 vertebra.
- Complete blood count showing anaemia, leucopenia.

- Raised ESR.
- Proteinuria and splenomegaly, indicating amyloidosis.
- All these features favor the diagnosis of multiple myeloma.

b)

1. **Secondary deposits in the bones/Metastatic** as suggested by:-

- a. Elderly lady with 6 months of general ill health, backache constipation.
- b. UMN paraplegia
- c. Raised ESR, anaemia.
- d. Partially collapsed T8 vertebra,

II. **lympho-proliferative disorders** (lymphoma, valdenstrom's macroglobulinaemia as suggested by:-

- a. Elderly lady with chronic ill health, backache and symptoms of hyperviscosity (dizziness).
- b. Skin lesions of hyperuricemia.
- c. Splenomegaly.
- d. Anaemia, leucopenia, raised ESR.

III. **Tuberculosis spine (Pott's disease):-**

- a. Emaciated patient .
- b. Backache, constipation.
- c. UMN paraplegia with collapsed T8 vertebrate.
- d. Anaemia, raised ESR.

c. **Investigations :-**

- CBC (anemia of chronic disease and rouleux formation)
- Serum electrolytes (hypercalcemia) , urea and creatinin levels
- Hypoalbuminemia and inc LDH
- Serum protein electrophoresis (monoclonal spikes in the beta and gamma globulin region)
- 24-hour urine immunofixation assay (presence of light chain in urine)
- Bone-marrow biopsy (>20% plasma cell)
- Serum beta-2 macroglobulin > 2.5 gm per dl (prognostic marker)
- Skeletal survey (lytic lesion in skull , spine , ribs and proximal leg bone)
- Gingival/rectal /renal biopsy for amyloidosis

Case No. 62:-

A 30 year old sewerage worker presented to the outpatient department with a history of high grade fever of one week duration. He also noticed anorexia and generalized aches and pains. One day he noticed dark coloured urine. He was taking no medications. He is a smoker of 10-12 cigarettes per day but there was no history of alcohol intake. On examination, he was having mild jaundice and tenderness in his right hypochondrium however; there was no visceromegaly, ascites or edema. Rest of the systemic examination was unremarkable. On investigations: Hb 11.8 g/dl, WBC $16 \times 10^9/L$, Platelets $128 \times 10^9/L$, bilirubin 3.2 mg/dl, AST 866 U/L, ALT 1760 U/L, ALP 395 U/L, gamma-GT 318 U/L, total proteins 6.4 g/dl and albumin 42 g/dl.

- What is the differential diagnosis?
- How will you investigate?
- What is the management plan?

a. differential diagnosis :-**1. leptospirosis (Weil's diseases).** The key points leading to this diagnosis are:-

- He is a sewerage worker. Sewerage system is inhabited by rodents, which are the main reservoir of leptospira.
- History of high grade fever with generalized aches and pains.
- History of hematuria.
- Raised bilirubin, deranged liver enzymes, raised WBC, low platelet.
- The combination of renal and liver derangement in a sewerage worker is highly suggestive of leptospirosis.

2. malaria :- Also reads as hepatic + renal**b. investigations:-**

- Urine C/E, may show protein, casts and red cells.
- Dark field examination of blood, may show leptospira.
- Culture of blood and urine for leptospira will be done, but it will take 1-6 weeks to show growth.
- ELISA or indirect haemagglutination for leptospira will be done.
- PCR for leptospira on blood will be done.
- Viral hepatitis serology, particularly HAV IgM, HEV IgM will be done. In addition to this HbsAg and anti HCV will be carried out.
- S/ urea and creatinine will be done to assess renal status.
- S/ electrolytes (potassium and sodium) will be checked.

c. Management :-

- Penicillin G, 6 million units, intravenously daily will be given.
- Strict fluid intake / output monitoring will be done.
- Fluid, electrolyte replacement will be carried out.
- Antipyretics and cold sponging will be done. In case of renal failure, dialysis will be carried out.

Case No. 63:-

A 27 year old man presented to accident and emergency department with a history of passage of loose motions for the last 5 days which were mixed with bright red blood and mucus. The frequency of blood was 10-12 day accompanied with fever and abdominal discomfort. There was no drug history. On further questioning he admitted passing loose motions mixed with blood and mucus, occasionally lasting for 2-3 days for the last 6 months but never had such bad attack before. One of his paternal uncles had similar problem but ultimately died of heart attack. On examination, he looked pale, toxic, dehydrated, temperature was 102°F pulse was 120/min, blood pressure was 110/70 mmHg. There was a soft systolic murmur over precordium. Abdomen was tender in left iliac fossa and left flank.

Investigations include CBC with P-film shows: Hb 7.8 g/dl (microcytic hypochromic with few tear drop cells), WCC $14.5 \times 10^9/L$ (Polys 82% L 14% Monocytes 2%, Eosinophils 2%) ESR 86 mm in 1st hour, sodium 140mmol/L, potassium 3.8 mmol/L, chloride 98 mmol/L, bicarbonate 24 mmol/L.

myelofibrosis

- What is the diagnosis?
- What is the differential diagnosis?
- How would you manage such case?

a) The most likely diagnosis is acute flare of ulcerative colitis:- as he is a young male patient with history of chronic diarrhea, haematochezia and abdominal pain. He is tender in left half of abdomen indicating predominant colon involvement. There is positive family history and fever, leukocytosis with predominant neutrophils and raised ESR.

b. Differential diagnosis :-

- crohn's disease
- Infective colitis.
- Ischemic colitis
- Antibiotic induced colitis

c. management :-**1. general management:-**

- Discontinue all oral intake for 24-48 hours
- TPN is indicated only in patients with poor nutritional status or if feeding cannot be re-instituted within 7-10 days
- All opioids and anti-cholinergic drugs should be discontinued
- Restore the circulatory volume by fluids and blood (if HCT < 25-28%)
- Stool examination for C-difficile, parasitis either by conventional bacterial culture , C-difficile toxin assay and ova and parasite examination by rapid multiplex PCR
- Serial plain abdominal radiograph or CT scan for colonic dilation
- DVT prophylaxis

2. specific management :-**i). corticosteroid therapy**

- Methylprednisolone 48-64mg or hydrocortisone 300mg is administered in four divided doses or by continuous infusion over 24 hours
- Hydrocortisone enema 100mg may be administered twice daily for treatment of urgency and tenemus
- 50-70% of patients achieve remission with systemic corticosteroid
- Once symptoms improvement has been occurred then oral fluid or reinstituted , systemic corticosteroid discontinue and started oral prednisolone
- Patients without significant improvement within 3-5 days of I/V corticosteroid should referred for surgery or considered anti-TNF therapies or cyclosporine

ii). Anti-TNF therapy :-

- Infusion of infliximab 5-10mg per kg is effective in treating severe colitis and patients who did not improved within 4-7 days of I/V corticosteroid

iii). Cyclosporine :-

- I/V cyclosporine 2-4mg/kg/day as a continuous infusion in treating severe colitis in patients who donot improve with in 4-7 days of I/V corticosteroid

iv). Surgical treatment :-

- Patients with severe disease who donot improved after corticosteroids , infliximab and cyclosporine then surgery is recommended

Case No. 64:-

A 35 years of age has noticed a worsening of his vision. New spectacles from the optician did not improve his sight. For the past one week, he is breathless. He gives history of headache, palpitations and diaphoresis off and on for the few past months. On examination pulse 98/min, regular respiratory rate 30/min, blood pressure 270/160 mmHg.

- What is the differential diagnosis?
- Name 5 investigations and justify how these will help to reach the most appropriate diagnosis?
- What is your management plan?

a. Differential diagnosis:-

- hypertensive retinopathy due to secondary hypertension
- pheochromocytoma (palpitation, headache, diaphoresis)
- primary hyper-aldoosteronism (hypernatremia, hypokalemia and metabolic alkalosis)

b. Investigations:-

- 24 hours urine collection will be done and the will be assayed for metanephrine and aldosterone. Metanephrines will be elevated in case of phaeochromocytoma (>2.2 ug/mg of creatinine), while aldosterone will be elevated in conn's syndrome (>20 ug in 24 hour urine sample). The metanephrine assay is 97% sensitive for detecting phaeochromocytoma.
- Serum sodium and potassium will be assayed. Patient with primary hyperaldosteronism (conn's) will have hypokalemia.
- Plasma renin levels, will be decreased in case of conn's and increased in renal artery stenosis and phaeochromocytoma.
- MRI abdomen will be ordered. On T2 weighted images, hyperintense adrenals are highly suggestive of pheochromocytoma.
- A whole body mibi scan to localize phaeochromocytoma.

c. Treatment :-

- He will be started on I/V nitroprusside (0.5 – 10ug/kg/min)
- Blood pressure monitoring will be done every 15 minutes.
- After reducing diastolic blood pressure to 110/120mmHg, intravenous b-blocker esmolol will be given before alpha-blocker as this may cause paradoxically rise in BP due to un-opposed alpha mediated vasoconstriction.
- ECG will be done hourly, to look for tachyarrhythmias.
- To prevent unopposed alpha medication, phenoxybenzamine will be given in a dose of 10mg every 12 hourly.
- In the long run he needs laparoscopic removal of the tumor. Pre-operatively, the blood pressure control with phenoxybenzamine and propranolol will be done.

Case No. 65:-

A 30 year old lady, who is K/C of SLE presented in a state of confusion in emergency department. History reveals that he was on prednisolone 60mg/day. Examination does not reveal any additional neurological signs. Investigations. Hb 10 gm%, platelet count 150000/cm, ESR 65 1st hour and urinary protein 500mg/24 hour.

- a. Give five differential diagnoses.
- b. Name five investigations with justifications.

a. Differential diagnosis :-

1. Lupus cerebritis, an important neurological manifestation of SLE.
2. Steroid induced psychosis.
3. uraemic encephalopathy .
4. Cerebrovascular accident (CVA), as it causes vasculitis. Vasculitis can lead to ischemia/infarction in the brain.
5. Electrolyte imbalance e.g. hyponatremia can lead to confusional state.

b. investigations:-

1. Cerebrospinal fluid (CSF) analysis, this will be carried out to look for any inflammatory disorder of brain e.g. cerebritis or meningitis. This could be either due to SLE itself or secondary to some infection as she is immunosuppressed due to her disease as well as steroid. CSF may show elevated proteins and WBC count.
2. CT/MRI brain, to look for any infarct, brain edema, or increased meningeal enhancement.
3. Renal functions; serum urea and creatinine so as to rule out uraemic encephalopathy.
4. Serum electrolytes; sodium and potassium to look for electrolyte imbalance.
5. CBC (WBC count to rule out septicemia)

Case No. 66:-

A 25 years old man weighing 60 kg who present with generalized anasarca. On examination, his jugular venous pressure is not raised; blood pressure in sitting position is 150/90mmHg. He has pallor; Hb is 10gm% and serum protein 4g%.

- a. Give four differential diagnoses.
- b. List the important investigations.
- c. Briefly outline the management plan.

a. Differential diagnosis :-***D/D of Generalized body swelling with out SOB***

1. Nephrotic syndrome
2. CKD
3. CLD
4. Malabsorption
5. Protein losing enteropathy

b. Investigations:-

1. Urine C/E; to look for haematuria, proteinuria.
2. 24 hour urinary proteins, in order to quantify proteinuria and spot urine , spot creatinine ratio .
3. Serum urea and creatinine, to assess glomerular filtration rate.
4. Serum electrolytes.
5. Serum albumin to document hypoalbuminemia. Fasting lipid profile .
6. Serum complement levels, as some glomerulonephritis like post-streptococcal causes hypocomplementemia.
7. Antibody profile e.g. ANA, ASO titer, ANCA will be advised, if above investigations support nephritic / nephritic syndrome.
8. Liver function tests including liver enzymes (ALT, AST) and PT , INR, for underlying liver disease.
9. Renal biopsy; so as to determine the cause of nephritic/nephritic syndrome according to the changes seen on light, electron microscopy and immunofluorescence.
10. Serum A/G ratio , usg abdomen for liver texture and kidney size .

c. management:-**general management :-**

- a. Strict control of hypertension is important. He will be advised ACE inhibitors / ARB's to control blood pressure as well as proteinuria.
- b. Dietary measures like low fat intake and protein intake of 1g/kg will be recommended.
- c. He will be advised and counselled regarding discontinuation of smoking.
- d. Anti-hyperlipidemic drugs e.g. HMG CO-A reductase inhibitors will be prescribed to control hyperlipidemia.
- e. Salt, fluid restriction will be advised.
- f. He will be given diuretics to reduce fluid overload.

g. For Proteinurea

- ACE / ARB (lower urine protein excretion by reducing glomerular capillary and antifibrotic effects)

h. For Edema

- Thiazid or loop diuretic

i. For Hyperlipidemia

- HMGCO-reductase inhibitor and for CKD gemfibrozol in combination with statin, fenofibrate or niacin with statin.

j. For Hypercoagulable state

- Warfarin

Specific treatment :-

- Specific treatment depends upon the results of kidney biopsy

Case No. 67:-

A 16 years old boy presents in the emergency department with history of repeated fits and unconsciousness for one day. On examination the blood pressure is 210/120 mmHg, temperature 38°C. there are no signs of meningeal irritation. CSF is normal. CT scan shows diffuse oedema of the brain. Serum creatinine 3.2mg%. Urine shows albumin++, RBC's++. Ultrasound reveals slightly edematous kidneys.

- a) What is the likely cause of the neurological features in this patient?
- b) How will you investigate this patient?
- c) What 4 steps would be important in the initial management of the patient?

a. Differential diagnosis of un-controlled hypertension in youngs:-**1. Reno-vascular diseases :-**

- Renal artery stenosis
- GN

2. Cardio-vascular diseases:-

- Coarctation of aorta

3. Endocrine hypertension :-

- Cushing syndrome
- Conn's syndrome
- Pheochromocytoma
- Thyroid dysfunction

4. connective tissue disorder :-

- SLE

5. ANC-positive vasculitis :-

b. investigation :-

- **Complete blood count with peripheral smear:** to look for anaemia, raised TLC, fragmented RBC's due to microangiopathic-haemolytic anaemia associated with hypertension.
- **Serum electrolyte:** sodium and potassium especially to rule out hyperkalemia.
- **Blood urea:** so as to assess the severity of renal failure.
- **Throat swab:** may show growth in case of post streptococcal glomerulonephritis.
- **ASO titer:** raised in post-streptococcal glomerulonephritis.
- **C3, C4 complement levels:** low in post-streptococcal glomerulonephritis, SLE, cryoglobulinemia.
- **ANCA** (anti-neutrophilic cytoplasmic antibodies) levels of c-ANCA are raised in Wegner's granulomatosis (another cause of nephritic syndrome)
- **ESR, ANA** (antinuclear antibodies); will be raised in SLE.
- **Serum IgA levels:** will be raised in IgA nephropathy.
- **Electrocardiogram** will be done to rule out any electrolyte disorder, associated with myocardial ischemia or arrhythmia.
- **24 hour urinary proteins:** to quantify proteinuria.
- **Renal biopsy:** if the patient does not respond to medication or have evidence of multisystem vasculitis.

c. management:-

1. general management:-

- As he has fits, is unconscious, so he needs to be resuscitated.
- Maintenance of airway is crucial. Airway will be passed. He will be kept in semiprone position. Fits will be controlled by giving him diazepam (10mg) followed by phenytoin 18-20 mg/kg I/VV.

2. Specific management :-

- To treat underlying glomerulonephritis, depending on the results of renal biopsy, he will be given steroids.
- Treat the blood pressure as a hypertensive emergency protocol.

Case No. 68:-

A 20 years old lady presents with history of anorexia, weight loss, low grade fever and cough for the last 6 months. She is also having diarrhea and abdominal pain alternating with constipation for the last 4 months. Examination revealed tenderness in right iliac fossa and hepatosplenomegaly. Examination of CVS is unremarkable.

- a. What is the most clinical diagnosis?
- b. Give four investigations with possible findings and justifications.
- c. How will you manage this case?

a.

The most likely clinical diagnosis is disseminated tuberculosis (miliary). She has symptoms and signs suggesting pulmonary, intestinal, lymph nodes and visceral involvement.

b. investigation:-

1. Complete blood count with ESR; will show normochromic, normocytic anaemia with raised ESR.
2. Chest x-ray; may show hilar/mediastinal adenopathy, upper lobe consolidation / cavitation / fibrosis and pleural effusion.
3. Excision biopsy followed by histopathological examination of cervical lymph nodes; this may show typical cavitating granulomas.
4. CT scans of the abdomen; may show intra-abdominal lymph nodes, matted gut loops, ascites, visceral involvement.

c. management :-**1. general management :-**

- nutritional status will be improved, by giving her balanced diet.
- Her close contacts should be investigated for latent tuberculosis, by mantoux test and if needed, will be given isoniazid (5mg/kg) daily for 9 months.

2. specific management :-

- She will be started on anti-tuberculous therapy (ATT). Four drug regimen; Isoniazid (5mg/kg), rifampicin (10mg/kg), pyrazinamide. (15-25mg/kg) and ethambutol (5-20mg/kg) will be given. This will be continued for up to 2 months then 2 drug regimen (isoniazide and rifampicin) will be continued for 7-10 months (total 9-12 months).

Case No. 69:-

A 60 years old male presented emergency with right side weakness of one week duration and blurring of vision in both eyes for 1 day. On examination he has a plethoric appearance with signs of upper motor neurons lesion on right side. Fundoscopy revealed dilated retinal veins. His haemoglobin is 19.4 G/dl. Renal function, lipid profile and LFT's were normal.

- a. What is the diagnosis and what is the pathogenesis of his eye lesions?
- b. List five renal cause of this condition.
- c. List five lab tests which you will order in this patient.

a).

- The diagnosis is secondary polycythemia (hyperviscous and hypercoagulable state) This condition is more common in males.
- Patient has signs and symptoms of hyper viscosity and hypercoagulability (retinal vein thrombosis and cerebral infarct).
- He has plethoric appearance.
- He has raised hemoglobin (19.4 g/dl).

PATHOGENESIS OF EYE LESIONS:-

Polycythemia due to raised red cell mass is characterized by raised viscosity and hypercoagulability. Hyperviscosity leads to blurring of vision, besides other symptoms (headache, tinnitus and pruritus after hot bath) while hypercoagulability can cause thrombosis at multiple sites, like this patient has retinal vein thrombosis as indicated by dilated retinal veins on fundoscopy and cerebral infarct

b).

Renal causes of this condition include:-

- 1 Hypernephroma
- 2 Polycystic kidney disease.
- 3 Wilm's tumor.
- 4 Hydronephrosis.
- 5 Renal transplantation.

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C.

- 1 CBC: Pack cell volume more than 55% is diagnostic to rule out spurious polycythemia.
- 2 White cell count and platelets counts; raised in case of polycythemia rubra vera.
- 3 CT/MRI head; to document cerebral infarct.
- 4 Arterial blood gases, to look for hypoxia, an important cause of secondary polycythemia.
- 5 Renal CT scan; to rule out renal cause of polycythemia.

Case No. 70:-

A 30 year old lady was found deeply unconscious in her room. She had no significant problems in the past. Twelve hours before she was seen in hostel mess taking dinner.

- a. List five possible causes of her condition.
- b. What five investigations would you request for?
- c. Give five initial management steps that you would take in the emergency room?

a).

- 1 Drug over dose (Poisoning , narcotic , diazepam and salicylates)
- 2 Head injury.
- 3 Metabolic cause:
 - a. Hypoglycemia.
 - b. Electrolyte imbalance (hyponatremia, hypokalemia)
- 4 Neurological cause:
 - a. Subarachnoid haemorrhage
 - b. Post-seizure coma
- 5 Septicemia (secondary to gastrointestinal / genitourinary infection)

b).

- 1 Drug screen (narcotics, diazepam, salicylates, etc.)
- 2 Blood sugar level, to rule out hypoglycemia.
- 3 Serum Electrolytes (sodium, potassium, calcium) serum urea and creatinine, to rule out fluid, electrolyte imbalance.
- 4 CT head to rule out trauma, sub-arachnoid haemorrhage.
- 5 Blood cultures, for septicemia.

c. management steps :-

- 1 I will start with monitoring of her vital signs (pulse, B.P, respiratory rate and temperature). Further management will proceed by maintaining these vitals. Any evidence of hypotension, brady/ tachycardia, tachypnoea, hypo/hyperthermia will be looked for and managed accordingly.
- 2 Fluids will be started intravenously. Nature of fluids will be determined according to the vitals. Saline will be given if she has hypotension, while dextrose water in case of hypoglycemia.
- 3 Calories, through nasogastric tube will be started.
- 4 Appropriate antibiotics will be started after taking blood cultures, if there is any evidence of infection.
- 5 Care of bladder (catheterization), bowel and skin will be started in the emergency.

Case No. 71:-

A man of 40 year complained of weight gain, fatigue and vomiting of one week duration. On examination B.P is 155/105 mmHg with peri-orbital puffiness. His BUN 85 mg/dl, s/creatinine is 9 mg/dl, urine analysis revealed proteinuria of 2+, RBC casts and leucocytes. Routine check-up by his physician a month earlier revealed normal B.P and lab tests.

- What is your diagnosis? Give reasons.
- List four serological tests you like to do.

a).

- The diagnosis in this patient is acute renal failure due to acute nephritic syndrome.
 - ❖ He had all the features of acute nephritis syndrome :-
 - ❖ Haematuria – RBC casts on urinalysis suggesting glomerular involvement.
 - ❖ Hypertension – B.P 150/105 mmHg.
 - ❖ Oedema (peri-orbital puffiness and history of weight gain)
- Uraemia – raised BUN (85mg/dl) and serum creatinine (9 mg/dl)
- A month earlier he had normal B.P and labs, thus suggesting acute onset of renal failure. Her symptoms of acute uraemia are vomiting, fatigue and fluid overload.

Causes of acute nephritic syndrome :-**1. Infections**

- PSGN

2. ANCA-positive vasculitis

- GPA (wegner's granulomatosis syndrome)

3. Connective tissue disorders

- SLE

b. investigations :-

- Anti-streptolysin-O titre, elevated in post-streptococcal nephritis, commonest cause of acute nephritic syndrome.
- Anti-neutrophilic cytoplasmic antibody (ANCA), present in vasculitis, which also cause acute nephritic syndrome.
- Anti-glomerular basement membrane (anti-GBM), seen in good pasture's syndrome, one of the cause of acute nephritis.
- Anti-nuclear antibody (ANA), present in systemic lupus erythematosus (SLE), in which renal involvement is seen in one third of the patients.

Case No. 72:-

A 22 year old man who has extensive soft tissue injuries because he was beaten, presents with passage of low quantity of urine and peri-orbital puffiness around eyes.

- a. What is the possible diagnosis?
- b. List five other causes which can give rise to similar presentation.
- c. How would you like to treat this patient with respect to his urinary problems?

a).

The patient has developed acute renal failure due to rhabdomyolysis. Individual with multiple soft tissue injuries is liable to develop necrosis of skeletal muscle, leading to myoglobinuria. Oliguria and peri-orbital puffiness suggests the acute renal failure.

b).

- 1 Drug overdose e.g. MDMA (Ecstasy) poisoning, statin group of drugs.
- 2 Prolonged immobilization.
- 3 Hypothermia
- 4 Neuroleptic malignant syndrome (NMS)
- 5 Status epilepticus.

c).

- He needs vigorous fluid replacement.
- At least 4-6 L/day has to be given. But at the same time, careful monitoring for fluid overload is to be done. Intake / output record monitoring will be carried out.
- Force diuresis by Mannitol (100mg/day) will be given as a diuretic.
- Urine alkalization (two ampules of NaHCO_3 in each liter of 0.5% normal saline) will be given.
- Any evidence of hyperkalemia on (ECG, lab report) will be treated with Calcium gluconate, Insulin/glucose, Beta-2 agonist nebulization.
- If he is still Oliguric, and starts developing pulmonary edema &/or hyperkalemia, then hemodialysis will be done.

Case No. 73:-

A 22 years old female presents with confusion, oliguria and melena, she developed fever, arthralgia and abdominal pain, one week back. On examination she was confused and had pallor, jaundice and petechial rash. Her neurological examination showed left sided facial palsy of lower motor neuron type. He examination showed Hb 8.0 gm/dl, TLC 10,000/mm³, platelets 95,000/mm³, bilirubin 3.2mg/dl, ALT 40 I.U. peripheral film showed fragmented RBC's and increased reticulocytes. Urine examination showed mid proteinuria and BUN was 60 mg/dl. Her PT and APTT were normal.

- a. What is the most likely diagnosis?
- b. Give three likely differential diagnoses you would consider?
- c. What other investigation would you carry out?
- d. How will you treat this case?

a).

The most likely diagnosis is **thrombotic thrombocytopenic purpura** as she has:

- CNS symptoms (confusion, left sided facial palsy)
- Renal involvement (oliguria, mild proteinuria, deranged renal function tests)
- Micro-angiopathic haemolytic anemia (decreased Hb fragmented RBC and increased reticulocytes on peripheral smear)
- Thrombocytopenia (malena, petechial rash, platelet count 95,000/mm³).

b).

- Hemolytic uremic syndrome (micro-angiopathic hemolytic anaemia, thrombocytopenia, prominent renal impairment, CNS involvement is less likely).
- Disseminated intravascular coagulation (microangiopathic hemolytic anaemia, thrombocytopenia, but PT and APTT are prolonged)
- Vasculitis/CTDS (young female patient with arthralgia, microangiopathic hemolytic anaemia, thrombocytopenia, renal and neurological impairment).

c). investigations:-

- Direct and indirect bilirubin, indirect bilirubin will be elevated as compared to direct bilirubin.
- Serum LDH, will be markedly elevated.
- Coomb's test, will be negative.
- Serum fibrinogen, will be normal.
- Fibrin degradation products (FDP), are usually not elevated.
- Absent or reduced levels of ADAMTS 13 (von-will brand cleaving protease) done.
- Renal biopsy, may show thrombosis of capillaries and small arteries, associated renal cortical inflammation and infarction; more prominent in HUS, as compared to TTP.
- ENA profile

d).

- 1 She will be treated with large volume plasmapheresis
- 2 60-80 ml/kg of plasma will be removed urgently and replaced with fresh frozen plasma. This will be continued until patient has improved.
- 3 In case of inadequate response, prednisolone and aspirin (325mg/tds) will be tried, in addition to plasmaphoresis.
- 4 Other options available in resistant cases are splenectomy and cyclophosphamide.

Case No. 74:-

A 60 year old gentleman presents with episodic loss of consciousness for the last 3 months and there is no residual damage. There is no history of fits.

- Give five conditions which you would consider in your differential diagnosis?
- What 6 investigations would you do to reach a diagnosis with justification for each?

a). Differential diagnosis of episodic loss of consciousness without fits and neurological deficit:-

- Vertebrobasilar transient ischemic attack (TIA)
- Cardiac arrhythmia, (strokes-adams syndrome)
- Aortic stenosis.
- Dysautonomia (may be due to diabetes, drugs or may be part of shy-dragger syndrome).
- Autonomic fits. (Epileptic drop attacks).

b). investigations:-

- Electrocardiogram:-** may show any evidence of arrhythmia, myocardial ischemia, left ventricular hypertrophy. 24 hours ambulatory ECG recording can be done for monitoring of arrhythmias. ECG response to posture, valsalva and deep breathing to diagnose autonomic insufficiency will be done.
- Carotid Doppler:-** vessels to determine any evidence of advanced atherosclerosis, and if required 4-vessel angiogram can be carried out to determine the cerebral vascular status.
- Transthoracic echocardiogram,** to look for aortic stenosis or cardiogenic source of thromboembolism.
- MRI brain** to look for any signs of brain stem ischemic disorder.
- Serum biochemistry:-**
 - Fasting glucose, as hypoglycemia can cause unconsciousness while hyperglycemia leads to autonomic insufficiency. Blood urea and creatinine as chronic renal failure causes autonomic insufficiency.
- Tilt-table testing,**
 - To rule out neuro-cardiogenic syncope.
 - This test allows to determine dysautonomia.
 - Pharmacologically, this test can be done by using isoprenaline.

Case No. 75:-

A 52 years old socially active lady started to feel undue tiredness for the last three months. She attributed this to overwork and decided to cut down on her activities. One month ago, she started to get breathlessness and consulted a doctor who ordered a chest x-ray and ECG. These were normal and she was reassured. One week ago she started to bleed from gums and noticed some spots on her legs. In the past she had enteric fever 2 years previously. She takes analgesics for her headaches. On examination; bruises on legs were noticed. Auscultation of heart revealed a grade II mid systolic murmur. Liver and spleen not palpable. Investigations: Hb .4 g/dl, TLC 1800/cmm with 19% polys. ESR 76 mm/1st hour. Urea 5.6 mmol/L, glucose 6 mmol/L.

- What is the likely diagnosis?
- What other points would you like to elaborate in the history?
- What investigations will you carry out?
- Discuss the management of this patient.

a).

Considering her symptoms of fatigue, dyspnea and bleeding from gums, pallor on examination with no hepatosplenomegaly, decreasing haemoglobin and TLC, the most likely diagnosis is Aplastic Anaemia.

D/D of Aplastic Anaemia is PNH:-

b. Essential of diagnosis :-

- History of fever, cough, sputum, chest pain, burning micturition, pain abdomen (evidence of infection in body)
- History of bleeding from any other site.
- Bone pains.
- History of drug intake and herbal medications .
- History of radiation exposure.
- History of exposure to insecticide.

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- History of exposure to other chemical agents.
- History of joint pain, skin rash.
- Family history of similar disorder.

c. investigations:-

- CBC : Pancytopenia .
- Peripheral smear to look for leucoerythroblastic picture in case of marrow infiltration.
- USG abdomen to rule out hepatosplenomegaly / intra-abdominal lymph nodes.
- Bone marrow trephine biopsy will reveal hypo-cellular marrow with no abnormal cells.

d. Management of Aplastic anemia :-

1. General management :-

- Control of infections with appropriate antibiotics
- Control of temperature by anti-pyretics
- Good hydration
- Good nursing barrier
- Red blood transfusion
- Erythroid or myeloid growth factors

2. Specific management :-

- If patient's age <40 years (allogenic bonemarrow transplantation)
- If patients's age >40 years (ATG 40mg/kg/day + cyclosporine 6mg /kg/day)
- If trilineage defects (thrombopoitin mimetic agents ELTROMBOPAG is added to cyclosporine)

Note :-

ATG (risk of serum sickness) can be given with corticosteroid 1mg/kg/day , tapered over 2 weeks

Case No. 76:-

A 25 years old man is admitted with a three days history of facial edema, dyspnea and mental confusion. On examination, he was pale looking JVP raised 5cm, pulse 115/min and regular, blood pressure 180/125 mmHg. Examination of fundi revealed AV nipping and vessel tortuosity. In the respiratory systems, extensive bilateral crepitations were auscultated. Other systems were normal. Hb 8g/dl, WBC 15,000, retics 4.0%, urea 250mg%, plasma sodium 124 mEq/L, potassium 5.8mEq/L.

- What are the three possible diagnoses?
- What are three most important problems in this case and how would you treat them?
- Give two causes for his anaemia.

a).Hypertensive emergency :-

- Hypertensive nephropathy
- Hypertensive retinopathy
- Micro-angiopathic hemolytic anemia
- Acute GN

b).

- Hyperkalemia
- Hypertension leading to renal failure, volume overload and anaemia due to microangiopathic haemolysis.
- Uraemic encephalopathy. For the treatment of hyperkalemia, he will be given I/V calcium gluconate, nebulized salbutamol, insulin in dextrose water and I/V bicarbonate. Intravenous frusemide will also be given. This will help to reduce hyperkalemia as well as hypertension. I/V diuretic will also reduce the volume overload. Further control of blood pressure will be achieved by the use of intravenous nitroprusside. Treatment of hypertension will help to prevent microangiopathic hemolysis as well. While all these measures will be carried out, dialysis will be planned as a part of renal replacement therapy. It will help to resolve all problems, that is hyperkalemia, hypertension, volume overload and uremic encephalopathy.

c).

- Normochromic normocytic anaemia due to erythropoietin deficiency secondary to renal failure.
- Microangiopathic hemolytic anaemia due to uncontrolled hypertension.

Case No. 77:-

A 21 year old unmarried girl is brought to the emergency department in coma. She is sweating, afebrile and tachypnoic with fine crackles at the lung bases. She generalized tonic clonic fit, which responded promptly to diazepam. Her Hb is 13.9 gm/dl, TLC $11 \times 10^9/L$, serum K 3 mmol/L and serum bicarbonate 10 mmol/L.

- What is the most likely diagnosis?
- What are three other conditions you would consider in your differential diagnosis?
- What four investigations would you carry out?
- Name two treatment modalities for this patient.

a).

The most likely diagnosis is Salicylate poisoning. Features suggesting this diagnosis are:-

- Unmarried, young female.
- Tachypnoic, sweating with B/L extensor plantar response.
- Afebrile, so infective cause is less likely.
- Normal electrolytes and calcium.
- Low serum bicarbonate suggesting metabolic acidosis.

b). differential diagnosis of salicylate poisoning:-

- Diabetic ketoacidosis, acidosis in a young patient should raise the suspicion.
- Poisoning with tricyclic antidepressants, amphetamines can also present with metabolic acidosis.
- Uremic acidosis; i.e. due to renal failure she has developed metabolic acidosis and encephalopathy.

c).

- Arterial blood gases to look at PCO₂, which will be low in compensatory respiratory alkalosis and arterial pH, which will be low due to metabolic acidosis (mixed respiratory alkalosis and metabolic acidosis)
- Blood sugar level, if elevated will be followed by serum / urine ketones to confirm diabetic ketoacidosis.
- Drug screening may show elevated salicylate levels.
- Serum urea and creatinine; for renal failure.

d).

Medical treatment:-

- 1 After obtaining history, gastric lavage with administration of activated charcoal will be done.
- 2 Metabolic acidosis will be treated with I/V sodium bicarbonate.
- 3 If the levels of salicylate in serum are between 400-700 mg/dl, urinary alkalinization will be done to promote renal salicylate excretion.
- 4 If salicylate levels are more than 700mg/dl, hemodialysis will be done.
- 5 Psychiatric management will be offered by counseling, education and referred of the patient to psychiatrist after she is stable.

Case No. 78:-

A 62 years old man was brought to emergency with history of malaise, fatigue and weight loss for 2 weeks. His wife said that he had been confused and drowsy and also had generalized convulsion 1 day back. He was a smoker and had smoked 20 cigarettes per day. There was no history of any drug intake. On examination he was drowsy and disoriented. Temperature was 37.8°C pulse 126 beats/min and supine B.P was 110/70. His erect systolic B.P was 80/60 mmHg. General physical and systemic examination was unremarkable. Investigations included Hb 11.0 g/dl, WCC 6.5 10⁹/L, 145 mg/dl (random), plasma osmolality 250 mosmol/kg, calcification and miliary shadowing throughout both lung fields.

- What are the two likely diagnosis?
- What three further investigations would you carry out?
- How will you manage the patient?

a). key points:-

- Fatigue, weight loss.
- Confused with generalized fits.
- Postural hypotension
- Increased ESR
- Hyponatremia, hyperkalemia, metabolic acidosis
- Pleural calcification and B/L pulmonary miliary shadowing

The two likely diagnoses are:-

- Miliary tuberculosis with tuberculous adrenalitis leading to Addisonian Crisis.
- Bronchogenic carcinoma with metastases to adrenal glands, causing Addisonian Crisis.

b).**Short synacthen test:-**

- Bronchoscopy, bronchoalveolar lavage** followed by lung biopsy. Lavage will be checked for AFB by ZN staining and culture. It will also be subjected to assays for malignant cells. Histological examination of biopsy specimen will establish the diagnosis.
- CT brain with contrast** to rule-out any infective / malignant process involving the brain.

c).

- After drawing blood samples for cortisol , give hydrocortisone 100-300mg I/V and saline.
- Hydrocortisone (50-100mg) will be continued every 6 hours I/V for the 1st day.
- Broad spectrum antibiotics after taking blood and urine cultures.
- Start anti-tuberculosis drugs, while carrying out the required investigations.
- If the disease is malignant then carry out palliative care, with consultation from the oncologist.

Case No. 79:-

A 58 years old bank executive, smoker, known hypertension taking enalapril 5mg/day, suddenly vomited and had double vision while sitting in his office, he also noticed that he has difficulty in swallowing, worse with liquids than solids. On examination, pulse 84/min, BP 170/100, heart sounds were normal and chest was clear. On right side he had ptosis and constricted pupil. Finger nose test is impaired on right side. Temperature sense in right arm and leg were intact but lost on the left side.

- a. What is the most likely diagnosis?
- b. What investigation you will carry out and why?

a). Most likely diagnosis is posterior circulation stroke (vertebro-basilar insufficiency) / lateral medullary syndrome / Wallenberg syndrome
essential of diagnosis :-

- Middle aged male
- Smoker
- Hypertensive
- Vertigo, vomiting, diplopia, dysphagia,
- Increased BP
- Ipsilateral horner's syndrome
- Ipsilateral loss of pain and temperature of face
- Contralateral loss of pain and temperature of limbs

b. Investigations :-

- MRI brain, as the lesion involves brain stem (medulla) which is not visualized on CT scan properly. MRI brain will be a preferable modality in this patient to look for evidence of ischemia or hemorrhage.
- After confirmation of stroke the investigations will be directed to look for the underlying cause. **These will include:-**
 - 1) Carotid duplex studies followed by MR angiography or conventional angiography.
 - 2) ECG, echocardiogram, chest x-ray.
 - 3) Holter monitoring if ECG suggests arrhythmia.
 - 4) Fasting lipid profile
 - 5) Serum homocysteine levels

- 6) Complete blood count to look for polycythemia, anaemia, raised TLC for any evidence of infection, thrombophilia.
- 7) Blood glucose.
- 8) Serological tests of syphilis if required.
- 9) ESR if any evidence of vasculitis present.
- 10) Blood cultures for endocarditis, if above investigations are inconclusive. If still no underlying evidence is found. Then work for hypercoagulable states (protein C, S antithrombin III, factor V leiden deficiency) will be done.

✓
Case No. 80:-

60 year old man k/C of T2DM presented with history of blurring of vision of right eye for 7 days O/E restricted extra-ocular movements of his right eye in all visual pursuit with dilated pupil and conjunctival congestion, further neurological examination revealed that impaired sensation in V1 and V2 distribution of Vth cranial nerve and his nasopharynx examination shows some growth

a: what is likely diagnosis ?

b: what is treatment options ?

A : Mucormycosis leading to right sided cavernous sinus thrombosis

B: i). Reversal of predisposing conditions

ii). Prompt antifungal treatment (amphotericin B 5mg / kg + caspofungin)

3 + V₁ + V₂ .

Differential diagnosis of multiple cranial nerve palsies:-

3, 4, 6 (3+2)

- 3, 4, 5, 6, 7, 8, 9, 10, 11, 12
- III , IV , VI , and VI , V₂ component of V cranial nerves (cavernous sinus thrombosis)
 - III , IV , VI and V₁ of component of V cranial nerves (SOF superior orbital fissure syndrome)
 - III , IV , VI , V₁ of component of V cranial nerves and II cranial nerve (orbital apex syndrome)

Causes of multiple cranial nerve palsies:-

- 2, 3, 4, 6, V₁, 10
1. Infections
 2. Demyelination
 3. Vasculitis / CTDS (mononeuritic multiplex)

If cause is not known called Tolosa Hunt syndrome

Case No. 81:-

25 year old boy presented with history of high grade fever on alternative day and painful joints for last three months , O/E cervical lymphadenopathy and splenomegaly , lab data shows anemia , TLC count 60,000 with predominant neutrophils and serum ferritin levels 3560mg/ml

- a: what is likely diagnosis ?
- b: what type of arthritis is present in this disease ?
- c: what is jaccoud's arthritis ?
- d: what is treatment ?

a: Adult onset still disease

b: destructive arthritis

c: defined has persistent peri-articular inflammation , which results in destruction of integrity of joint capsule and supporting ligaments called jaccoud's arthritis

d: i. NSAIDS

ii). Corticosteroids

iii). Targeting IL-1 (anakinra or canakinumab)

iv). IL-6 (tocilizumab)

Case No. 82:-

25 year old female with history of painful joints and worsening of abdominal distention for last 3months O/E mild icteric conjunctiva , liver is edged blow right costal margin with total span 18cm , soft in consistency , smooth surface and painful lab data shows HB 10gm% , ANA is positive , C3 C4 complement level reduced and anticardiolipin antibodies are detected .

- a: what is likely diagnosis ?**
b: what is likely cause of diagnosis ?
c: what are likely treatment options ?

- a: Budd-chiarri syndrome
 b: SLE / APLS

c:

1). Ascites should be treated with salt restriction and diuretics

2). Treat the underlying disorders

3). To maintain the patency of hepatic vein by

- TIPS with polytetrafluoroethylen-covered stent
- Balloon angioplasty with metallic stent

4). To prevent propagation of clot :-

- Thrombolytic therapy
- Anticoagulations (choice / duration / type of anticoagulation depends upon the underlying disorders)

5). Liver transplantation :-